Noise and health of children

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September 2000

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Summary

Noise and children

The world of the child is becoming noisier and noisier. Compared to the mid-fifties environmental noise levels (sources such as road traffic, aircraft) increased substantially, causing higher noise levels during day- and night-time at home, at school and during out- and indoors leisure time activities. Also, children spend increasingly more time in situations with (many) other children, such as in day care institutions and kindergartens, with high noise levels due to a combination of loud voices, loud toys, and bad acoustics. In an unknown percentage of households, nowadays television or audio-equipment is turned on for the whole day, thus creating an everlasting noisy environment for the child. Children may be more annoyed or otherwise adversely affected by noise than adults, in part because they possess less well-developed coping responses, and are often less able to control their environments. It is unknown to which extent aggressive behaviour, helplessness, and hyper-activity are (in part) a consequence of the everlasting noise exposure of the young and older child.

In contrast to the extent of noise exposure of children is the extent of research into the effects of noise exposure on their health. It is largely unknown which adverse noise-induced effects occur in children and also at which levels these effects start to occur. Usually environmental noise regulations based on exposure effect relationships for adults are assumed to be applicable to the child as well.

This report gives an overview of the adverse effects of noise exposure on the health of children. The overview is based on data obtained from the literature. In this summary the possible effects of noise on children’s health are discussed, with children classified according to age. Health is assumed to include biological (physiological, somatic), psychological, social and emotional aspects.

Fetus

Three types of possible effects due to high noise levels during gestation of the mother have been considered:

- Hearing impairment assessed in epidemiological surveys in which the noise exposure of the pregnant mother was the decisive factor with respect to noise load. In the surveys, audiology was performed when the children reached school age
- Effects associated with birth outcomes: low birth weight, gestational age and growth retardation
- Abnormalities of the baby originated during pregnancy (teratogenesis).

The information available on noise-induced effects on the fetus shows hearing impairment associated with exposure to high occupational noise levels during gestation. On-going research
indicates that also growth retardation of the child is associated with extensive occupational noise exposure of the pregnant mother. It cannot be excluded, but is seems unlikely, that environmental noise causes fetal abnormalities. Overall, the studies on the effect of environmental noise on the fetus have been hampered by serious methodological limitations, both in terms of assessment of noise exposure and effect, and failure to control for known determinants of the effects under investigation.

**Pre-term and full-term babies**

There are substantial differences between the pre-term and full-term baby. Pre-term babies must cope with their environment with immature organ systems. The auditory, visual and central nervous systems are the last systems to mature. These last stages occur, in part, during the time the pre-term child is in the incubator or neonatal intensive care unit (NICU). Also, the sleep-wake patterns differ markedly among pre-term and full-term infants.

It has been recognised for long that high noise levels exist in the NICU and incubators, the environment in which the premature baby usually lives for shorter or longer periods up to months. Measurements in the NICU have shown equivalent sound levels from 60 to 90 dB(A) with maximal levels of very loud events up to 120 dB(A). The equivalent sound levels in the incubator are 60 to 75 dB(A), and when ports of the incubator are closed maximal sound levels up to 100 dB(A) occur.

Four types of adverse noise-induced effects on the pre-term baby have been considered:

- Hearing impairment
- Sleep disturbance
- Somatic effects
- Effects on auditory perception and emotional development

**Hearing impairment**

In premature babies the hearing organ is still developing after birth. Taking into account the extra vulnerability for hearing impairment during development of the hearing organ, higher levels of NICU and incubator noise is to our opinion able to produce noise-induced hearing impairment in pre-term babies. However, there is no research carried out which could support this statement.

**Sleep disturbance**

Noise events in the NICU and incubators are sufficiently loud to have an effect on sleep, either by awakening the infant or by changing the sleep state. Pre-term infants who have difficulty maintaining stable behavioural states experience the same or greater sleep disruption as do term infants to similar stimuli.

**Somatic effects**

Through the increased number of awakenings and associated crying the effect of noise in the incubator and the NICU is a potential cause of hypoxemia and source of neonatal morbidity.
Fluctuations in arterial oxygen tension, blood pressure and intracranial pressure may contribute to hypoxic brain damage. The decrease in oxygen saturation of blood can affect all the vital organs. The infant residing in the NICU or incubator can experience acute effects many times in the period of rapid brain growth. Potential consequences include increased risk of weakened vessel walls in the cerebral vasculature. Unfortunately in-depth research on this subject is lacking.

**Auditory perception and emotional development**

Current knowledge strongly suggests that stimulation provided by the auditory environment plays a role in the emotional development and in the development of auditory perception of the baby. The sound quality in the NICU and incubator is reduced, since speech and other relevant sounds are masked. It is also difficult for infants in an incubator to localise the origin of air-borne sounds and these sounds contain less higher frequency components. This impaired sound quality implies that the pre-term infant may have difficulties in making fine discriminations with respect to (the intonation of) the voice of the mother and caretakers. The possible emotional implications for the pre-term baby at a later stage are unknown.

**Pre-school and school children**

The following effects have been considered:

- Hearing impairment
- Effects on sleep
- Somatic effects
- Psycho-social effects

Noise-induced somatic effects (such as on blood pressure and hormone levels) can best be considered as part of a stress response of children to their noisy environment. Psychological and cognitive processes also play a role in this stress response of children. Therefore, somatic (physiological) results should be considered together with psychological outcomes to give an overall insight in the problem.

It is an important question whether prolonged noise exposure results in increasingly adverse effects on children or whether those exposed for longer periods adapt to the situation with effects disappearing after a while. The relevance for health and development is clear if the effect or effects studied have a permanent nature and do not disappear. On the other hand, if a survey shows that adaptation of the measured effect variables occurs, it is unsure what the price of these temporary effects is on other variables that were not measured. For instance, if a real life study on the effect of noise exposure on psycho-physiological stress-related variables (blood pressure, cognitive performance) shows that the child adapts to the noise situation at school, it is uncertain what the price is on other functions such as aggressive behaviour, unless that variable was measured as well.

**Hearing impairment**

The investigations undertaken so far show that environmental noise exposure does not have an
effect on hearing threshold levels of children, with the exception of exposure to noise from extremely low flying military aircraft. However, taken into account the very high noise levels present during 24 hours in mega-cities, research in this area might show hearing impairment in children associated with these very high noise exposures. Given the high noise emissions of specific toys and equipment, some noisy activities may impair the hearing of children. Potential sources of hearing impairment in children are: toddlers noisy toys, fire-crackers, tractors and other agriculture machines, snow mobiles, shooting equipment, power tools, musical instruments, walk- and disc-mans. Although hearing impairment has been reported in isolated cases, the results of large-scale hearing surveys with school children fail to show increases in hearing impairment attributable to noise exposure.

**Effects on sleep**

There are only a few observations with respect to the effects of noise during sleep on sleep parameters of children. The few test results do not contradict the hypothesis that – in analogy with physiological reactions in the waking state - physiological responses occur in children at a lower event level than in adults. On the other hand, even if the child is awake, as measured by sleep EEG, it usually does not produce a behavioural response, such as pressing a button. In particular during REM sleep, noise events of sufficient intensity are able to cause EEG awakenings in children. During the last third of the night, in which REM sleep is predominant, children under experimental conditions show 50% EEG awakenings due to noise signals with maximal levels of up to 95 dB(A) above threshold. Although children exposed at home may show less awakenings, this is an important finding, because of the necessity of REM sleep for memory consolidation. The few test results obtained so far give an indication that noise events in the first part of sleep (evening-time) do have less impact on sleep of children than noise events in the early morning. Since sleep is very important to health and development of children, much more research is needed to obtain a more detailed insight in possible adverse noise-induced effects.

**Somatic effects**

Only one, older, cross-sectional study showed unambiguously that environmental (aircraft) noise exposure is associated with an increase in (rested) systolic and diastolic blood pressure. In the more recent Munich aircraft noise study, noise-induced increase in stress hormone (epinephrine and nor-epinephrine) levels could be established. In all other studies, covering aircraft and road traffic noise, differences in physiological parameters of noise-exposed children and children not exposed to high levels of noise effects were either not statistically significant, absent or it could not be excluded that intervening variables could (partly) explain these differences.

With respect to physiological adaptation, the data presented on road traffic noise show an increase with age in the differences in blood pressure between noise-exposed and not exposed children (no adaptation), whereas all data on aircraft noise exposure show decreasing differences with duration of exposure (adaptation). If possible effect-modifying factors would not have played a role, this would imply that children physiologically adapt to a certain degree to aircraft noise, but not to road traffic noise. As pointed out earlier, this does not imply that the child also adapts to aircraft noise exposure in all other aspects or that long-term consequences are absent.
Psycho-social effects

Some of the adverse effects of environmental noise on children may be caused indirectly by noise effects on their caretakers. Studies show significant interruptions and lost teaching time in schools with high traffic noise levels. Also, teachers in these schools report noise annoyance and irritation due to the noise and dissatisfaction with their working situation. Parents in noisier homes, with most of the noises generated indoors, are less responsive to their children than those in quiet homes. Perhaps the speech patterns of the parents, teaching and demonstrating behaviour or engagement in cognitive related activities (reading aloud) is adversely impacted by noise. There are no studies available on behaviour of parents living in homes with high environmental noise levels.

Most studies on the psychological effects of noise exposure on children are focussed on aspects of cognition. Nearly all of these studies selected children in specific noisy and quiet schools as study and reference populations. School children, with a long-term exposure to high levels of traffic noise from either aircraft, road or railway traffic, do show impairments in performing cognitive tasks under quiet conditions. The best-documented noise effect is that on reading acquisition. Several studies have found indications of a negative relationship between long-term noise exposure and reading acquisition (measured under quiet conditions). There are fewer studies of noise effects on other aspects of cognitive processing, such as long term memory, attention, and motivation of children. The studies which have examined possible links between noise exposure and attention deficits among children show varying results. Several investigators found an effect of long-term noise exposure on the performance of a task (a visual search task or an auditory sustained attention task), while other researchers did not. Of interest is the finding that a visual coding task was performed better under acute noise conditions by children attending noisy schools than by children attending quiet schools, whereas they did worse on the task when performing it under quiet conditions. These and other findings suggest that attention deficits related to long term noise exposure in children occur because children learn how to ignore auditory stimuli (gate out distraction) as a way to cope with long-term noise exposure. Unfortunately, this tuning out process may over-generalise so that children learn to tune out not only noise, but also relevant other auditory signals, such as speech.

Some studies showed that children highly exposed to environmental noise for prolonged periods of time are less motivated when placed in situations where task performance is dependent on persistence. These motivational deficits in children related to long term noise exposure have been considered in the light of the learned helplessness theory. Prolonged exposure to uncontrollable stimuli has been shown across a wide variety of conditions, including noise, to induce feelings and behaviours indicative of helplessness. As the child continues to struggle unsuccessfully with an uncontrollable stimulus, it eventually learns that it is helpless to do anything about the situation, as manifested by feelings of hopelessness and reduced persistence. Like in adults, this effect is strongly mediated by personal characteristics.
Teenagers

There is a nearly complete lack of research into the somatic, psycho-physiological and behavioural effects of noise on teenagers, nor are there studies on noise-induced sleep disturbance of subjects of this age group. The only noise effect in teenagers to which a lot of studies have been devoted is noise-induced hearing impairment.

Hearing impairment

The potential sources of hearing impairment mentioned for schoolchildren (noisy toys, fire-crackers, tractors and other agriculture machines, snow mobiles, hunting equipment, power tools, musical instruments, walk- and disc-mans) may also impair the hearing of teenagers. In addition, it is not unlikely that noise levels in boom-cars and (under helmets) of motor cycles cause noise-induced hearing impairment in teenagers.

A part of the older teenagers is already employed. The relationships presented in ISO 1999 (1990) about noise-induced hearing impairment and noise exposure show that during the first 10 years of exposure hearing impairment at the most affected frequency (4000 Hz) is only somewhat less than after a life time exposure. Therefore, to preserve good hearing in case technical noise abatement measures are not taken, it is important that teenagers are instructed to use personal hearing protection from the beginning they start being exposed to high noise levels, not only at work but already at technical schools and polytechnics. The extent of hearing impairment in teenagers caused by occupational noise exposure and exposure at technical schools and polytechnics is unknown.

Most of the studies on hearing impairment in teenagers concern the effect on hearing threshold levels of exposure to popmusic in discotheques, at popconcerts and house parties and when listening through headphones. Many of these studies have been limited to the assessment of the degree of hearing impairment in teenagers, without trying to specify exposure effect relationships. In a study about the relationship of hearing threshold levels and exposure to pop-music through headphones, it was made plausible that the model given in ISO 1999 for occupational exposure of adults also holds, albeit with a slight adaptation, for this type of exposure and for teenagers. Whether the model also applies to the much more irregular exposures of teenagers to pop-music at pop-concerts, discos and dance halls, is unknown.

Although noise-induced hearing impairment among teenagers has been reported in isolated cases, a comparison of the present distributions of hearing threshold levels of young populations with those distributions 30 years ago fails to show increases in this distribution.
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1 Introduction

1.1 Framework

This report gives an overview of the adverse effects of noise on the health of children. It is the basis for a contribution to a report to the European Commission, Directorate for Public Health and Safety at Work (DG V/F), in the framework of the first part of a project ‘Health effects of noise on children and perception of the risk of noise’, coordinated by National Institute of Public Health (Denmark).

1.2 Contents of the report

The report contains an overview of the adverse effects of noise exposure on health of children, based on data obtained by a literature search. It also gives to a certain extent models of how these effects are generated. General information on effects of noise on human beings has been obtained from: Hobson, 1989; Passchier-Vermeer, 1993; Health Council of the Netherlands, 1994; Berglund et al., 1995; Shaw, 1996; Thompson, 1996; Morrell, 1997; IEH, 1997; WHO, 1999; Passchier-Vermeer et al., 2000. With respect to effects of noise exposure on children, general information was obtained from: Mills, 1975; De Joy, 1983; Passchier-Vermeer, 1991, 1993; Horne, 1992; Evans et al., 1993, 1997. Publications already available at the Institute after an extensive literature search in 1992 have been reconsidered with respect to information about noise effects on the health of children. Also a literature has been performed to find publications from 1990 about the subject under consideration. Where appropriate reports and publications cited in the information already at hand have been consulted. In the various chapters the possible effects of noise on children’s health and wellbeing are discussed with children classified according to age. In appendix A, the characterisation of noise exposure is given. Effects of environmental noise exposure on adults are given in appendix B.

1.3 Characterisation of child

A child is a developing, growing, maturing human being. In considering the effects of noise on children’s health, the following general features of human health should be considered in the light of development, growth and maturation:

- Biological (physiological, somatic)
- Psychological
- Social
- Emotional
To understand fully the effects on children of noise in their daily environment, it is not sufficient to consider the effects of noise on each of these features separately, but also to take into account possible interactions between the effects on these features.

1.4 Methodologies

In considering the literature about the effects of noise on children the following methodological aspects have been taken into account:

- have effects been “proven” in real life situations of children or have they “only” been observed under experimental test conditions.
  The impact of noise on children can best be assessed by a methodological strategy that combines the results of experimental (laboratory) studies and epidemiological surveys. Laboratory studies allow us to assess adverse (temporary) effects of noise on children in a chosen specified environment and with the possibility to exclude or manage effect-modifying factors. They have the disadvantage that it is uncertain whether the effects observed also occur in real life. Epidemiological surveys help to establish whether particular effects found in the laboratory or derived from laboratory studies do indeed occur in real life and to which extent. Epidemiological real life studies have the disadvantage that it is difficult to isolate the effects of a particular environmental factor of the living environment, such as environmental noise, on the health (and behaviour) of children or adults.

- is there a plausible model for the underlying mechanism, in which an observed effect of noise on children and the observed direction of the effect fits.
  With the exception of explorative studies, it is imperative to base laboratory studies as well as epidemiological surveys on a detailed model with as a hypothesis a “cause – effect” chain from noise exposure to adverse noise-induced effect on children and with possible effect-modifying factors. Also, the design of the study has to be such that it allows the hypothesis to be tested. In principle, only associations between variables are assessed. It depends, among other things, on the plausibility and strength of the model whether it is reasonable to assume the associations to have a causal relationship.

- do specific functions of children adapt to noise exposure in the course of time or not and what are possible long-term consequences of noise exposure, irrespective of the adaptation of the functions studied.
  It is an important question whether prolonged noise exposure results in increasingly adverse effects on children or whether those exposed for longer periods adapt to the situation with effects disappearing after a while. Evidently, the relevance for health and development should be taken into account if the effect or effects studied have a permanent nature and do not disappear. On the other hand, if a survey shows that adaptation of the measured effect variables occurs, it is unsure what the price of these temporary effects is on other variables that were not measured. For instance, if a real life study on the effect of noise exposure on psycho-
physiological stress-related variables (blood pressure, cognitive performance) shows that the child adapts to the noise situation at school, it is uncertain what the price is on other functions such as aggressive behaviour, unless that variable was measured as well. A method to study adaptation is by longitudinal intervention surveys, in which variables are studied in the course of time and in which the child acts as its own control. The Munich Airport Noise and Schoolchildren Study is such an investigation: measurements on children in the course of time (before and after the closing of the old airport and the opening of the new airport) and intervention by closing the old airport and opening the new airport (Hygge et al., 1996; Evans et al., 1998). Even in a study with such a design, a careful interpretation of the test results with respect to adaptation is necessary: not only changes in the noise environment may have an effect on the variables studied, but also changes in other factors of the environment. Another aspect that needs a careful analysis of the longitudinal study results is attrition bias: children with the largest adverse effects may not turn up at re-examinations, thus leaving for longitudinal conclusions a sub-group which is not representative for the original study population (Cohen et al., 1981).
2 Effects on the fetus (unborn child)

2.1 Introduction

Three types of possible effects due to high noise levels during gestation of the mother are considered:
- Hearing impairment, assessed in epidemiological surveys in which the noise exposure of the pregnant mother was the decisive factor with respect to noise load. In the surveys, audiometry was performed when the children reached school age
- Effects associated with birth outcomes
- Abnormalities of the baby originated during pregnancy (teratogenesis).

2.2 Hearing impairment

The human cochlea and peripheral sensory end organs complete their normal physical development by 24 weeks of gestation, whereas the maturation of the auditory pathways of the central nervous system occurs up to 42 weeks of gestation. From animal experiments it is known that during this process of maturation, the hearing organ has an extra sensitivity for impairments (Gerhardt, 1990).

During fetal development gradually the so-called tonotopic organisation takes place. Initially during gestation, only low frequency sounds are registered by the fetus. In this process the hair cells of the cochlea at the beginning of the basilar membrane near the oval window are involved, whereas the hair cells at the end of the basilar membrane of the fetus do not transmit any signals. This is in contrast to the adult human cochlea, in which for high frequency sounds the hair cells at the beginning of the basilar membrane are involved and low frequency sounds are perceived through hair cells located at the end of the basilar membrane. As the membrane and other structures mature, the low frequency sounds are registered farther and farther along the membrane, and the hair cells near the oval window begin to register higher and higher frequencies. This is called shifting tonotopic organisation. To date it appears that the various perception centers of the brain undergo the same shifts in tonotopic organisation over the course of development. In the human fetus, these changes are in process by week 12 of gestation and continue during the early weeks after term birth.

The shift in tonotopic organisation in the fetus implies, also taking into account the various sound pathways and the attenuation of airborne sound by the abdomen of the pregnant woman, that in the first months of gestation the unborn baby perceives internal and external low frequency noise only. In the last period of normal pregnancy the unborn baby starts to perceive the (higher pitched) voice of the mother.

Two epidemiological studies (Daniel et al., 1982; Lalande et al., 1986) showed high frequency hearing impairment in children of women exposed during pregnancy to occupational noise with equivalent sound levels exceeding 85 dB(A). Lalande shows an increase in impairment if low
frequency components are present in the occupational noise environment of the pregnant woman. No studies have been performed on the possible relationship between environmental noise exposure of pregnant women and hearing loss in young children. At present new techniques such as measurements of otoacoustic emissions are used on a routine basis in mass screening programs in new-borns. This gives the opportunity to study such a relationship. With respect to the underlying mechanism of the hearing impairment, two possible mechanisms may be involved in the destruction of the inner hair cells of the developing cochlea: directly through noise exposure of the fetus or as a result of a decreased blood supply to the uterus due to stress related reactions of the pregnant mother. Taken into account the extra effect of low frequency noise on the hearing impairment in the children studied, it seems more likely that noise directly affects the hearing organ of the unborn child.

2.3 Low birth weight, premature birth and growth retardation

Several studies have examined the association between environmental noise (in most studies aircraft noise) and low birth weight and premature birth. In view of data from older studies (Ando et al., 1973; Rehm et al., 1978; Knipschild et al., 1981; Schell, 1981), high levels of aircraft noise to which pregnant women are exposed, may give a small increase in percentage of babies with a low birth weight (less than 2500 gr). In a more recent study of 200 Taiwanese women, noise exposure was measured by personal noise dosimeters on three occasions during pregnancy (Wu et al., 1996). Birth weight turned out not to be related to noise exposure, after adjustment for social class, smoking and alcohol use, maternal weight gain in pregnancy, gender of the child and duration of pregnancy.

A recent publication of the ELSPAC Study (European Longitudinal Study of Pregnancy And Childhood) performed in the Czech Republic looked at associations between the occupational situation in the period before and during pregnancy and health of babies (Hrubá et al., 1999). The occupational situation was assessed by a written questionnaire filled in by the mothers in the period after delivery and there were no (noise exposure) measurements carried out at the work place. The population studied consisted of 3897 women. It was shown that the odds ratio for growth retardation of the baby was 1.9 for those women exposed to occupational noise each full working day during and before pregnancy. Also, head circumference of the babies of these noise-exposed women was statistically significant smaller than the head circumference of the babies of the other women. Smoking turned out to be highly correlated with occupational noise exposure. Unfortunately, the publication does not specify whether the results for growth retardation and head circumference were controlled for the modifying effect of smoking.

2.4 Fetal abnormality (teratogenesis)

Without consideration of the investigations mentioned in the previous two sections, ten investigations (each from before 1994) have been carried out to establish effects of occupational or environmental noise exposure of pregnant women on congenital defects of babies (for references,
see Passchier-Vermeer, 1993). Most investigations usually are not based on a model that specifies the underlying mechanisms, nor do most of them show any statistically significant effects of noise exposure. Incidentally a statistically significant noise exposure effect is shown. An inventory study on congenital malformations around Los Angeles International Airport showed a significantly higher rate of birth defects in black people exposed to high levels of aircraft noise compared with unexposed black people (Jones et al., 1978). However, the survey was criticised because of the lack of completeness and accuracy of the birth defects data and because potential confounding factors were not taken into account. Also, Edmonds et al. (1979) found that one birth defect (spina bifida with hydrocephalus) out of 17 defects studied was statistically significant associated with exposure to high aircraft noise levels. This result about spina bifida and aircraft noise exposure was not reproduced, however, in his case-control study at the same airport on a larger set of data.

Overall, the studies on the effect of environmental and occupational noise on the fetus have been hampered by serious methodological limitations, both in terms of assessment of noise exposure and effect, and failure to control for known determinants of the effects under investigation.

2.5 Conclusion

The information available on noise-induced effects on the fetus shows hearing impairment caused by occupational noise during gestation and possibly growth retardation. With respect to growth retardation further results from the ELSPAC study have to be awaited. It cannot be excluded, but is seems unlikely, that environmental noise causes fetal abnormalities.
3 Pre-term and full-term babies

3.1 Introduction

There are substantial differences between the pre-term and full-term baby. Pre-term babies must cope with their environment with more immature organ systems. The auditory, visual and central nervous systems are the last systems to mature. These last stages occur, in part, during the time the pre-term child is in the incubator or neonatal intensive care unit (NICU). Also, the sleep-wake organisation differs markedly among pre-term and full-term infants (Holditch-Davis et al., 1987). As this organisation is an important indicator of the functioning of the brain, these findings suggest that the central nervous system of the premature and of the full-term infant function differently. The sleep-wake pattern of an infant not only reflects endogenous processes but also the ongoing response of the baby to environmental stimulation. There are also strong indications that the overall mother-infant relationship differs for premature and full-term infants (Oehler, 1993). These neurobehavioral differences have implications for the later development of pre-term babies. The differences in brain functioning probably alter specific perceptual, cognitive, and emotional processes, rather than overall intelligence (Philbin, 1996; Blackburn, 1998).

It has been recognised for long that high noise levels exist in neonatal intensive care units and in incubators, the environment in which the premature baby usually lives for shorter or longer periods up to months (Gaedecke et al., 1969). During the last 30 years measurements in the NICU have shown equivalent sound levels from 60 to 90 dB(A) with maximal levels of very loud events up to 120 dB(A) (Benini et al., 1996; Guimaraes et al., 1996; Philbin et al., 1999; Brezinka et al., 1997; Gray et al., 1998; Raghu Raman, 1997; Robertson et al., 1998). Noise in the NICU is from air flow, oxygen monitoring devices, ventilators, monitor alarms, printers, telephones, personnel communication and laughter, door closings. The incubator gives poor protection for room noise. Typically, the equivalent sound level in the incubator is 60 to 75 dB(A), and when ports of the incubator are closed maximal sound levels up to 100 dB(A) occur. Most noisy events are associated with various activities of the staff: closing of doors, diaper pails, incubator ports, and drawers, laughter, rubbish disposal, and conversations carried on across the length of the nursery (Long et al., 1980; AAP, 1997). In some hospitals special programs have been carried out to instruct personnel about quieter behaviour in the NICU.

3.2 Hearing impairment

In premature babies the hearing organ is still developing after birth. Taking into account the extra vulnerability for hearing impairment during maturation of the hearing organ, higher levels of NICU and incubator noise are to my opinion able to produce noise-induced hearing impairment in pre-term babies. However, there is has no research been carried out that could support this statement (Gerhardt, 1990; AAP, 1997).
3.3 Sleep disturbance

Noise events in NICU and incubators are sufficiently loud to have an effect on sleep, either by awakening the infant or by changing the sleep state (Gaedecke et al., 1969; Strauch et al., 1993; Smeczuk, 1967; Lota, 1992). Pre-term infants who have difficulty maintaining stable behavioural states experience the same or greater sleep disruption as do term infants to similar stimuli. A study was devoted to the effect of wearing mini ear muffs on behavioural and physiological responses of premature babies in incubators (Zahr et al., 1995). When earmuffs were worn pre-term infants had statistically significant higher mean oxygen saturation levels and smaller fluctuations in these levels. Furthermore, these infants had less behavioural state changes, spent more time in quiet sleep state and had longer bouts in the sleep state. Therefore, noise in NICU and incubators does have an adverse effect on sleep of pre-term babies.

Peculiar study results were published by Ando et al. (1977). They studied reactions of 71 full-term babies to aircraft noise and to music with the same frequency content and time history as the aircraft noise by means of sleep EEG and plethysmography. With respect to aircraft noise, babies of mothers who lived in the vicinity of the airport during pregnancy showed much less EEG and plethysmographic reactions than babies of mothers who moved to the area during the last months of pregnancy, after delivery or who lived in an area without aircraft noise exposure. The differences between these two groups of babies in plethysmographic responses were less for music than for aircraft noise. It is difficult to postulate the underlying mechanism that explains these results.

3.4 Somatic effects

In figure 1 a recording is given, taken from a one week old pre-term male infant in an incubator, of heart rate, respiratory wave, transcutaneous oxygen tension (TcPO$_2$), and intracranial pressure (ICP). Sudden loud noises cause agitation, and crying, which usually lead to an increase in heart rate and respiratory wave, decrease in oxygen tension and an increase in intracranial pressure (Long et al., 1980). Through the increased number of awakenings and associated crying the effect of noise in the incubator and NICU should be considered a potential cause of hypoxemia and source of neonatal morbidity (Long et al., 1980). Fluctuations in arterial oxygen tension, blood pressure and intracranial pressure may contribute to hypoxic brain damage and intracranial haemorrhage. The decrease in oxygen saturation of blood can affect all the vital organs. The infant residing in the NICU or incubator can experience many such acute effects in the period of rapid brain growth. Potential consequences include increased risk of weakened vessel walls in the cerebral vasculature.

3.5 Other effects

Current knowledge strongly suggests that stimulation provided by the auditory environment plays a role in the emotional development and in the development of auditory perception of the baby.
From an acoustical point of view, the following observations should be made. The sound quality in NICU and incubator is reduced, since speech and other relevant sounds are masked. It is also difficult for infants in an incubator to localise the origin of air-borne sounds and these sounds contain less higher frequency components. This impaired sound quality implies that the pre-term infant may have difficulties in making fine discriminations with respect to the intonation of the voice of the mother and caretakers. The possible emotional implications for the pre-term baby at a later stage have not been studied.

3.6 Conclusion

Noise in the NICU and incubator impairs the sleep of pre-term babies and is, at high levels, able to induce hearing impairment. It is a potential cause of hypoxemia and potential source of neonatal morbidity. Further, noise exposure and masking of specific sounds may have consequences on the emotional and behavioural development of pre-term babies.
4 Pre-school and school children

4.1 Introduction

There are strong indications that the environment in which children nowadays live is increasing in noisiness (Sargent et al., 1993). Compared to the mid-fifties environmental noise levels (sources: road traffic, aircraft) increased substantially, causing higher noise levels during day- and night-time at home, at school and during leisure time activities. Also, children spend increasingly more time in situations with (many) other children, such as in day care institutions and kindergartens, with high noise levels due to a combination of loud voices and bad acoustics. In an unknown percentage of households, nowadays television or radio is turned on for the whole day, thus creating an everlasting noisy environment for the child. Children may be more annoyed or otherwise adversely affected by noise than adults, in part because they possess less well-developed coping responses, and are often less able to control their environments. It is unknown to which extent aggressive behaviour, helplessness, hyper-activity are (in part) a consequence of the persistent noise exposure of the young and older child.

4.2 Hearing impairment

Young children may be more susceptible to noise-induced hearing impairment than adults. This is made plausible in the figures 2a and 2b. Figure 2a gives the result of experiments with mice, an animal with the same physiology of the hearing organ as humans (Passchier-Vermeer, 1991). It gives the effect of exposure to very high noise levels as a function of age of the mouse. The effect has been assessed by counting hair cell loss in exposed mice killed after noise exposure or by measuring cochlear microphonics in living mice. The effect is presented relative to the effect at the age of four days. If we convert the developmental stage of mice to that of human children (figure 2b), it is obvious that susceptibility for hearing impairment in pre-school and school children is greater than that of adults, at least in case of very high noise exposures. Whether this is also applicable to real life noise exposures of children is unknown. In Appendix B it has been made plausible that exposure to environmental and leisure time noise with $L_{Aeq,24h}$ values below 70 dB(A) does not cause hearing impairment in the large majority of adults (over 95%), even in case of life time exposure. If the young child is more vulnerable in acquiring noise-induced hearing impairment at the lower exposure levels, the observation threshold will be below 70 dB(A). Regarding noise exposure relevant for children, this implies that a large proportion of young children have on a regular basis noise exposures that are above this observation threshold (for noise exposure data relevant for children, see Passchier-Vermeer, 1993).

Several large scale audiometric investigations of school age children have been carried out in the seventies and eighties. These investigations showed that the hearing threshold levels of boys and girls up to an age of about 10 years are about the same. At ages over 10 years, the percentage of
boys with hearing threshold levels exceeding a certain value (in the order of 20 dB) is larger than that of girls. This difference was attributed to higher noise exposures of boys (Axelsson et al., 1981). Whether this suggestion is correct or not is a matter of debate, since there are no scientific data to support or contradict the statement. In section 5.2 hearing threshold levels of teenagers are discussed. There it is stated that by far the largest part of the distributions of the hearing threshold levels of teenagers and of the adult population did not change during the last 25 years or so. This implies that it is quite unlikely that there is in general a degradation in the hearing threshold levels of still younger age groups. This does not contradict the possibility of noise-induced hearing impairment in a small portion of the general population of pre-school and schoolchildren. Apart from the ‘normal’ noise exposures of children in their living environment (household noises, radio, TV, voices), specific higher noise exposures of the young child can be classified as to environmental noise (examples of sources are dense road traffic and aircraft) and to noise during noisy activities (examples of sources are noisy toys, pop-music through headphones). The possible impact of these exposures on the hearing threshold levels of children is discussed below.

4.2.1 Environmental noise exposure

Several epidemiological surveys in developed countries with children living in highly noise exposed areas included in the test population, did not show any effect on hearing threshold levels from exposures to environmental noise (Carter et al., 1975; Andres et al., 1975; Fisch, 1981). On the other hand Chen et al. (1993) and Ising et al. (1991) did show such effects. Chen and Chen determined statistically significant higher hearing threshold levels of sixth grade elementary school children (11 – 12 years) exposed to aircraft noise relative to a reference population. However, the mean hearing threshold levels of the noise-exposed as well as of the reference group over the whole frequency range were extremely high. This indicates that either audiometric testing was of a low quality or other factors have had an adverse effect on the hearing threshold levels of both groups. These unknown factors might then also explain the differences in hearing threshold levels observed. It is unlikely that the shifts in hearing threshold levels observed over the whole frequency range are due to noise exposure, since noise-induced hearing impairment involves the higher frequencies and low frequency hearing is usually not impaired. Ising et al. assessed hearing threshold levels of children exposed to noise from low-flying military aircraft. At one of the study locations, 9 – 13 years old children living under a so-called 75 m flight corridor had a statistically significant increase of 2 dB in mean hearing threshold levels (2000 to 8000 Hz) compared to children living under 150 m flight corridors.

4.2.2 Noisy activities

Toys, mentioned to be used by young children, such as squeaking toys, toy cars with sirens and toy pistols, are able to produce high peak sound pressure levels (Hellström et al., 1992; De Joy, 1983; Passchier-Vermeer, 1991). Whether exposure to such noises induces hearing impairment in very young children (toddlers) has not been examined in epidemiological surveys. Only fragmentary, an effect on a particular child has been reported (Brookhouser et al., 1992). Noise-induced hearing impairment largely depends on the number of exposures during shorter periods
of time, the years of exposure to noisy toys, the number of noisy activities in the course of years and the way activities are performed, and there is no knowledge about these factors for toddlers and young children. However, taking into account the high noise exposures and a possible extra susceptibility in acquiring noise-induced hearing impairment, in principle noise-induced hearing impairment in some toddlers due to loud toys should not be excluded.

In older children specific noise exposures may also have an impact on their hearing. High noise levels and noise-induced hearing impairment have been reported due to fire-crackers. Other sources documented to have caused noise-induced hearing impairment in highly exposed groups of school children are: tractors and other agriculture machines, snow mobiles, motor vehicles, hunting sports, power tools, musical instruments, and loud audio equipment (Fletcher, 1972; Fletcher et al., 1977; Dickinson et al., 1989; Razi et al., 1995). Considering the very high noise levels in so-called boom-cars, this situation should also be considered as a potential source of noise-induced hearing impairment, even for pre-school and school children.

With respect to loud audio equipment, children start listening to pop-music through headphones of walk- and disc-mans at a much lower age than formerly (Passchier-Vermeer, 1997), at least in the Netherlands. It is quite common in the Netherlands that children younger than 12 years nowadays use walk- and disc-mans with headphones to listen to pop-music on a daily basis. It has also been shown in a survey that the youngest children examined (12 to 15 years) turn the volume regulator of their walk- or disc-man to much higher settings than (older) teenagers (Passchier-Vermeer, 1997). There is a growing insight in the scientific community, based on results of surveys among pop-music listeners, that listening to pop-music through headphones induces and induced less hearing impairment in teenagers than expected and stated formerly (report of meeting 1999 in Munich to be published). However, if the total number of listening years and the listening levels increase, the future cumulative exposure in the course of years to pop-music through headphones of walk- and disc-mans would be a potential source of hearing impairment of children.

4.2.3 Conclusion

Environmental noise exposure, with exception of extremely low flying military aircraft, most probably does not impact the hearing of children. Some noisy activities may impair the hearing of specific groups of children. Potential sources of hearing impairment in children are: toddlers noisy toys, fire-crackers, tractors and other agriculture machines, snow mobiles, motor vehicles, hunting sports, power tools, musical instruments, walk- and disc-mans if used extensively by young children, and possibly boom-cars.

4.3 Effects on sleep

4.3.1 Sleep of children

Sleep is a recovery process that is essential for humans to function properly. In Appendix B various aspects of sleep are discussed. From the sleep EEG, two distinct phases of sleep are
distinguished. These phases are: NREM sleep and REM sleep, also called dream sleep (REM is rapid eye movement). NREM sleep covers four stages, stages 3 and 4 are called deep sleep (slow wave sleep: SWS) and stages 1 and 2 light sleep (in these stages the transition from SWS sleep to REM sleep or awakening occurs). In general, body restoration was assumed to occur mainly during NREM sleep and brain restoration mainly during REM sleep. However, recently it was shown that memory consolidation, as part of brain restoration, not only takes place during REM sleep, but also that SWS in the first part of the night contributes significantly to memory consolidation (Stickgold, 1998). There are essential physiological differences between NREM and REM sleep. During sleep, adults have sleep cycles of about 90 minutes in which REM and NREM sleep occur alternating (see figure B1 in Appendix B).

In figure 3 the time spent in the various wake sleep stages are given for human beings of various ages. For one week-old babies the average sleep length is 16 hours (with a standard deviation of 2 hr). After a year the average sleep length is 13 hours. Total sleep time declines almost entirely as a result of the decline of REM sleep from 8 hours at birth to 4 hours at one year. After about one year the sleep pattern of babies is about that of adults, be it that the cycle REM NREM is 60 to 70 minutes. Regular day-time naps disappear by about 5 years. Total amount of sleep decreases to 12 hours at 2 years, 10 hours at 5 years, 9 hours at six to seven years, 8.5 hours at 8-9 years, 8 hours at 10-11 years. The sleep cycle is 80 to 90 minutes for the average 5 years old child, REM sleep has then reached the adult proportion of 20 to 25%. SWS starts to occur at about 4 months, is about 50% of NREM at 1 year, and remains at this high level for several years. By age of 9 years SWS sleep is 22 to 28% of sleep time.

4.3.2 Noise effects

Only four publications are related to the effects of night-time noise on parameters of sleep of young children (5 to 12 years old).

Lukas (1972) studied 22 test subjects, among them 6 children (5 to 7 years of age). Subjects were exposed to noise from aircraft flyovers and sonic booms at times that the sleep EEG showed they were asleep in stage 3 and 4 (SWS sleep). Results are presented in figure 4. Reactions to a noise event were classified as “no awakening” as shown by the EEG, “awakening” according to the EEG but without a behavioural response (pressing a button) and “behavioural awakening”, awakening with a behavioural response. During SWS sleep children appear to be less easily awakened than adults and behavioural awakenings are nearly absent in situations in which adults, especially older people, do show with a behavioural response that they are awake.

Eberhardt (1988) studied by using EEG recordings, actimetry (body movements) and questionnaires the effects of road traffic noise on sleep of 13 children (aged 6 – 11 years). Eight children (group 1) lived in quiet surroundings and were exposed to on average 68 pre-recorded truck noises with maximal sound levels of 45, 55 and 65 dB(A) during several nights. Five children (group 2) lived along noisy streets. Exposures of these five children during several nights were lowered by double glazing of their bedroom (attenuation on average 11 dB(A)). Subjects from group 1 showed increased body movements (assessed 0 – 60 s after onset of noise): 9% at 45 dB(A), 5% at 55 dB(A) and 10% at 65 dB(A). Awakenings occurred during on average 0.2, 0.6
and 2.3 percent of the events respectively. One child showed 8.5% awakenings due to truck noise with a maximal level of 65 dB(A). There were no statistically significant changes in any of the EEG parameters studied, with the exception of an increase in intermittent wakefulness from 4 to 10 minutes under the noisiest exposure condition. After noisy nights, the children experienced that it took them longer to fall asleep, it happened more that they remembered to have been awake at night, and the reason for awakening was more often road traffic noise. After noisy nights they also rated their sleep quality lower and their feeling rested worse. The most substantial effect on sleep EEG of the noise reduction for group 2 was a reduction in sleep latency time with 7 minutes. Eberhardt considers children to be less sensitive to noise-induced awakenings and body movements than adults. He estimates that the same reactions in the sleep EEG in adults and children occur if the night-time noise exposure of the children is 10 dB(A) higher than the exposure of the adults.

Busby et al. (1985) studied 24 (8 – 12 years old) boys, 8 not medicated hyperactive boys, 8 medicated hyperactive boys and 8 not hyperactive boys. Sleep EEG’s were recorded in the sleep laboratory during 4 nights, the first two served as adaptation nights. The stimulus was a 1500 Hz tone with duration of 3 s (alternating 2 times on and off) with an increasing intensity (2 to 5 dB increment) presented during known stages of sleep, and administered through an insert earphone. Threshold values while awake were 28 dB. The maximal level that could be produced was 123 dB (which implies 95 dB above threshold). In the pooled subject data (no differences between groups were observed) the frequency of awakening was 4.5, 34, 50% for SWS, stage 2 and REM sleep respectively. The percentages of partial arousals, i.e. changes in EEG without awakening, and EEG awakening are given in figure 5 as a function of time of night. During the last part of the sleep period of the child, in which the child is most of the time in REM sleep, the stimulus was able to produce 50% awakenings, whereas this was 15% during the first part of the sleeping period, in which the child is most of the time in SWS sleep.

Semczuk (1967) examined the effect of acoustic stimuli on respiratory movements by thoraxographic registration in 50 children (age 5 to 7 years) and 100 adults during sleep (for the children from 21 h to 01 h). Children reacted with changes in the respiratory curves to acoustic stimuli of much lesser intensity (10 to 15 dB) than adults did. This might imply that the central nervous system of children is aroused more easily than that of adults.

4.3.3 Conclusion

The first conclusion is that there is very little research into the effects of noise during sleep period time on sleep parameters of children. The second observation is that, in contradiction to general belief, children react to night-time noises. The test results do not contradict the hypothesis that on average vegetative responses occur in children at a lower event level than in adults. On the other hand, even if the child is awake according to the sleep EEG, it usually does not produce a behavioural response, such as pressing a button. In particularly during REM sleep, noise events of sufficient intensity are able to cause EEG awakenings. During the last third of the night, in which REM sleep is predominant, children show 50% EEG awakenings due to noise signals with maximal levels of up to 95 dB(A) above threshold. This is an important finding if it is considered with a view on the necessity of REM sleep for memory consolidation. The few test results ob-
tained so far give an indication that evening-time noise events do have less impact on sleep of children than noise events in the early morning. It may also explain why parents usually think noise does not awake their child, because observations by parents are usually made during the evening and not during the early morning. Moreover, if children awake in the early morning and do not give a behavioural response before they fall asleep again, parents are not aware of this awakening. Since sleep is very important to health and development of children, much more research is needed to obtain a more detailed insight of possible adverse effects.

4.4 Stress-related somatic effects

4.4.1 Field studies

In table 1 an overview is given of the surveys on stress-related somatic effects in school children, in casu on the blood pressure and on neuro-endocrine indices of chronic stress of these children. In most instances also measures of cognitive performance were assessed (see section 4.5). In each survey, schools and children from specific school classes were selected. Children were then classified according to the exposure to a specific noise source (road traffic, aircraft) out- and inside the classroom. Usually young children attend schools in the close neighbourhood of their home and most likely there is a high correlation between noise exposure during class and exposure at home and while playing outside. Therefore the question remains whether noise-induced effects should be exclusively attributed to noise exposure during class, or whether exposures in other situations (at home) also had an impact on the effects observed. This reasoning seems also valid in case of cognitive effects of noise exposure on children. This is the more so, since sleep disturbance caused by night-time noise is able to impair memory reprocessing during sleep.
Table 1  Surveys on blood pressure and neuro-endocrine indices of chronic stress in school children

<table>
<thead>
<tr>
<th>Reference</th>
<th>Noise source and type of study</th>
<th>Time of measurements</th>
<th>Mean values (val) or mean differences (diff) between noisy and quiet classes or level of significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Karsdorf et al., 1968</td>
<td>Road traffic noise Cross-sectional study</td>
<td>During class hours</td>
<td>Age (years)</td>
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<td>16</td>
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<tr>
<td>Cohen et al., 1980</td>
<td>Aircraft noise Cross-sectional, first part of longitudinal study</td>
<td>Before start of school</td>
<td>Exp (years)</td>
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<td></td>
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<td>&gt; 4</td>
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<tr>
<td>Cohen et al., 1981</td>
<td>Aircraft noise Longitudinal study, first part see Cohen et al., 1980</td>
<td>Before start of school</td>
<td>No statistically significant effects (tested one-sided at a significance level of 0.05)</td>
</tr>
<tr>
<td>Lercher, 1992</td>
<td>Highway noise Cross-sectional study Various environmental factors</td>
<td>During class hours</td>
<td>Percentage of children with higher blood pressure and higher cholesterol level</td>
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<td></td>
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<td>Far from highway syst 26 dia 6 chol 62</td>
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<tr>
<td>Regecova et al., 1995</td>
<td>Road traffic noise Cross-sectional study</td>
<td>During class hours</td>
<td>Group (see text) val syst/diast (mm Hg)</td>
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<td>4</td>
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<tr>
<td>Evans et al., 1995</td>
<td>Aircraft noise Cross-sectional, first part of longitudinal study</td>
<td>Before start of school (resting blood pressure) and during class hours (difference between these measures is a measure of the reactivity of blood circulation)</td>
<td>Level of significance</td>
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<td>Resting diastolic -</td>
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<td></td>
<td>Reactivity diastolic -</td>
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<td></td>
<td></td>
<td></td>
<td>Epinephrine (+43%) +</td>
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<td>Cortisol (+4%) -</td>
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<tr>
<td>Evans et al., 1998</td>
<td>Aircraft noise Longitudinal study, first part see Evans et al., 1996</td>
<td>Before start of school (resting blood pressure)</td>
<td>Exp (years) syst/diast (mm Hg)</td>
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<td>0.5 – 1.5</td>
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Two early cross-sectional studies showed higher systolic and diastolic blood pressure in school children exposed to very high road traffic noise levels (Karsdorf et al., 1968) or very high aircraft noise levels at school (Cohen et al., 1980) than children not exposed or with minor exposure to these noise sources. Karsdorf et al. (1968) measured blood pressures of 13 to 16 years old secondary school children in the first five hours after beginning of class. The results show an increase with age in the (statistically significant) differences in systolic and diastolic blood pressure between noise exposed children and children not exposed to loud road traffic noise at school. Unfortunately, known effect-modifying factors (body weight, smoking, social class, diet, alcohol use) have not been taken into account. Therefore, it is largely unknown whether the actual noise exposure caused (all of) the effect reported. Cohen et al. (1980) measured rested blood pressure in advance of the beginning of school. His study shows unambiguously that rested blood pressure and noise exposure at school are associated. Cohen et al. (1981) re-examined children from the first investigation again one year later. Of the 262 children from the first investigation, only 163 took part in the second investigation. It turned out that a large proportion of the aircraft noise exposed children with higher blood pressure did not participate in the second investigation. The analysis of the attrition sample of the longitudinal study did not show any effect of noise exposure, testing session, or interactions between noise exposure and testing session on either systolic or diastolic blood pressure.

Lercher (Lercher, 1992) examined 796 school children living close to or far from highways. The study does not only consider noise exposure, but also other environmental factors, such as exposure to lead. The results are presented as percentages of children with a systolic blood pressure over 120 mm Hg, with a diastolic blood pressure over 80 mm Hg or with cholesterol levels over 176 mg/dl. Blood pressure measurements were mostly performed in the morning from 9 to 12 hours. The results observed are contradictory to the hypothesis of higher values in the higher noise exposed children, and this contradiction remains if effect-modifying factors are taken into account.

More recently Slovakian researchers studied 1542 3-7 year old children from kindergartens (Regecova et al., 1995). They estimated the road traffic noise exposures at the kindergartens and at the homes of the children. The children were classified according to these two noise exposures in four groups (road traffic noise with equivalent sound levels below or above 60 dB(A)): 1 quiet kindergarten and quiet home, 2 quiet kindergarten and noisy homes, 3 noisy kindergarten and quiet homes, 4 noisy kindergarten and noisy homes. Measurements on blood pressure and heart rate were performed in the morning (8.30 to 12.00 hours). The authors observed significantly higher systolic and diastolic blood pressure and lower heart rate in groups 3 and 4 compared to group 1 and 2, after control for age, weight, and height. The differences in mean systolic and diastolic blood pressures of the various groups were lower in the youngest age group and increased with age. Although the study is carefully designed, the possibility exist that social class can, in part, explain the differences observed (see also Lercher et al., 1998).

In the Munich airport study, schoolchildren were examined in the years Munich airport moved from one to another location (Hygge et al., 1996; Evans et al., 1998). One location was situated close to the ‘old’ airport and the other close to the ‘new’ airport. The cross-sectional part of the study showed a, not statistically significant (P = 0.08), higher systolic blood pressure in children highly exposed at school (Evans et al., 1995). Children were matched on socio-economic char-
acteristics. In the study also neuro-endocrine indices of chronic stress (urinary cortisol levels and levels of epinephrine and norepinephrine) were examined. Overnight resting levels of epinephrine and norepinephrine levels were significantly higher in the children exposed to aircraft noise at the old Munich airport in comparison to the control group. There were no differences in cortisol levels. After the move of the airport, overnight resting levels of epinephrine and norepinephrine levels rose significantly among children living under the flight paths of the new airport. There was, again, no effect on cortisol levels.

4.4.2 Conclusion

Only the cross-sectional study of Cohen et al. (1980) shows that aircraft noise exposure (as specified at school) is statistically significant associated with increase in systolic and diastolic blood pressure. In the Munich study, noise-induced increase in epinephrine and nor-epinephrine levels could be established. These results can best be considered as part of a stress response of children to their noisy (school) environment. Psychological and cognitive processes also play a role in this stress response of children. Therefore, somatic (physiological) results should be considered together with psychological outcomes to give an overall insight in the problem (see section 4.5). Amazingly, the surveys in which physiological as well as psychological variables have been studied never reported about the correlation between both sets of effect measures. Concerning adaptation, the data presented by Karsdorf and by Regecova on road traffic noise show an increase with age in the differences in blood pressure between noise-exposed and not exposed children (no adaption), whereas all data on aircraft noise exposure show decreasing differences with duration of exposure (adaptation). If possible effect-modifying factors would not have played a role, this would imply that children physiologically adapt to a certain degree to aircraft noise, but not to road traffic noise. As pointed out earlier, this does not imply that the child also adapts to aircraft noise exposure in all other aspects nor that long term consequences or other effects are therefore absent.

4.5 Psycho-social effects

4.5.1 Introduction

The main psycho-social effects of noise exposure in adults are annoyance and specific disturbances, such as speech interference and interference with other activities such as listening to radio or TV. Like in adults, also in children, raised background noise levels mask speech and interfere with speech perception, language acquisition and subvocalisation processes (Cohen et al., 1973). Studies in which school children were observed have shown that in classes with high levels of aircraft or railway noise children are distracted by intermittent noises. There are no large scale studies on environmental noise exposure and noise annoyance of children. Most studies on the psycho-social effects of noise exposure on children are focussed on aspects of cognition. Nearly all of these studies selected children in specific noisy and quiet schools as study and
Some of the adverse effects of environmental noise on children may be caused indirectly by noise effects on their caretakers. Studies show significant interruptions and lost teaching time in schools with high traffic noise levels (Bronzaft et al., 1975). Also, teachers in these schools report noise annoyance and irritation due to the noise and dissatisfaction with their working situation. Parents in noisier homes are less responsive to their children than those in quiet homes (Wachs, 1979; Wachs et al., 1991; Matheny et al., 1995). The three studies mentioned assessed interior noise with most of the noises generated in the homes. Perhaps the speech patterns of the parents, teaching and demonstrating behaviour or engagement in cognitive related activities (reading aloud) are adversely impacted by noise. There are no studies available on behaviour of parents living in homes with high environmental noise levels.

4.5.2 Research results

For over thirty years epidemiological studies have shown that school children, when exposed to high levels of traffic noise, do show impairments in performing cognitive tasks (railway noise: Bronzaft et al., 1975; aircraft noise: Cohen et al., 1980; road traffic noise: Karsdorf et al., 1968). The best documented noise effect is that on reading acquisition (Green et al., 1982; Evans, 1997; Evans et al., 2000). Close to twenty studies have found indications of a negative relationship between noise exposure and reading acquisition. There are fewer studies of noise effects on other aspects of cognitive processing, such as long term memory, attention, and motivation of children. The most ubiquitous memory effects occur when complex, semantic materials are probed: several studies on long term or acute noise exposure have found adverse effects of aircraft noise exposure on long term memory for complex, difficult materials. The dependency of differences in adverse noise effects on the complexity or difficulty of the memory task could be related to the distinction between implicit and explicit memory. Meis et al. (1998) found that both long term and acute aircraft noise interfered with long term recall but had no impact on word production. Implicit memory tasks like word production are resistant to distraction or divided attention, whereas explicit memory tasks such as recall are more vulnerable to interference. Long term noise exposure does not appear to have an effect on short term memory. The studies which have examined possible links between noise exposure and attentional deficits among children show different results. Several investigators found an effect of long term noise exposure on the performance of a visual search task or of an auditory sustained attention task, while other researchers did not. Various variables may moderate the relations between long term noise exposure and performance on a sustained attention task. Of interest is the finding that young children from noisy homes were less distracted by auditory signals during a visual matching task than children from quiet homes (Heft, 1979). It was also found that compared to children attending quiet schools a visual coding task was performed better under acute noise conditions by children attending noisy schools whereas they did worse on the task when performing it under quiet conditions (Hambrick-Dickson, 1986). These and other findings suggest that attentional deficits related to long term noise exposure in children occur since children learn how to ignore the noise.
auditory stimuli (gate out distraction) as a way to cope with chronic noise. Unfortunately this tuning out process may over-generalise so that children learn to tune out not only noise, but also relevant other auditory signals such as speech.

Some studies showed that children highly exposed to environmental noise for prolonged periods of time are less motivated when placed in situations where task performance is dependent on persistence. The motivational deficits in children related to long term noise exposure have been considered in the light of the learned helplessness theory. Prolonged exposure to uncontrollable stimuli has been shown across a wide variety of conditions, including noise, to induce feelings and behaviours indicative of helplessness. As the child continues to struggle unsuccessfully with an uncontrollable adverse stimulus, it eventually learns that it is helpless to do anything about the situation, as manifested by feelings of hopeless and reduced persistence. Like in adults, this effect is strongly mediated by personal characteristics of the child.

More recently two longitudinal studies were carried out (Evans et al., 1995, 1998; Hygge et al., 1996; Hygge et al., 1998; Haines et al., 1998). In the Munich airport study (a longitudinal intervention study), reading comprehension and long term memory were impaired in children around the old Munich airport and reading comprehension improved after the closing of the airport. At the same time, it deteriorated in children subjected to the aircraft noise exposure near the new Munich airport. Recently, in the UK a field study with annually repeated tests was carried out to assess whether the association between aircraft noise exposure and reading comprehension was mediated through sustained attention and whether it was confounded by social deprivation and language spoken at home (Haines et al., 1998). The 340 children that participated were aged about 9 to 10 years. They visited a school classified either as a high noise school ($L_{\text{eq,schoolhours}}$ over 66 dB(A)) or as a low noise school ($L_{\text{eq,schoolhours}}$ less than 57 dB(A)). There appeared to be a high correlation between noise at school and the aircraft noise exposure at home. The results show that on average reading comprehension of children attending the high noise schools was poorer at both measuring times compared with that of children from the low noise schools. Sustained attention, only measured at follow-up, was poorer in the children at the high noise schools than in the children at the low noise schools. Sustained attention did not play a significant role in the explanation of the relation between reading comprehension and aircraft noise exposure. However, if adjustments were made for age, main language spoken at home and social deprivation, the differences between children from high and low noise schools in reading comprehension failed significance.

4.5.3 Conclusion

Given the possible long-term consequences of cognitive effects in children, further research into the mechanisms and contributing factors is very much needed. In that research a link has to be made between the psychological and somatic effects.
5 Teenagers

5.1 Introduction

There is nearly a complete lack of research into the psycho-physiological and behavioural effects of noise on teenagers, nor are there studies on sleep disturbance on subjects of this age group. For instance, in the large TNO database (with data on annoyance and sleep disturbance of over 63 000 subjects), only 571 (0.9%) of the subjects are less than 18 years. The only noise effect in teenagers to which a lot of studies have been devoted is noise-induced hearing impairment. Most of these studies concern the effect on hearing threshold levels of exposure to popmusic through headphones, in discotheques, and at popconcerts. The next section considers this subject. Apart from popmusic exposure, teenagers may also be and have been exposed to potential damaging noise sources during noisy activities mentioned in the preceding section on school children. In addition, a part of the teenagers is exposed to loud noises during class at technical schools and polytechnics (Axelsson et al., 1981). A part of the older teenagers is already employed. If their exposure, measured on a yearly basis, exceeds an equivalent sound level during their workday of 75 dB(A), occupational noise-induced hearing impairment may occur. The relationships presented in ISO 1999 (1990) about noise-induced hearing impairment and noise exposure show that during the first 10 years of exposure hearing impairment at the most affected frequency (4000 Hz) is only somewhat less than after a life time exposure. Therefore, to preserve good hearing in case technical noise abatement measures are not taken, it is important that young persons are learned to use personal hearing protection from the very beginning they are exposed to high noise levels, not only at work but also at technical schools and polytechnics. The extent of hearing impairment in teenagers caused by occupational noise exposure and exposure at technical schools and polytechnics is unknown.

5.2 Hearing impairment

Many studies on hearing impairment of teenagers have been aiming at the assessment of the degree of hearing impairment in teenagers without trying to specify exposure effect relationships (Hetu et al., 1995). The specification of relationships is difficult indeed, since there are many variations in exposure parameters. Usually, it is very difficult to obtain sufficient quantitative data about the exposure in the past and present (Babisch et al., 1989; Struwe et al., 1996). E.g. with respect to popmusic exposure at popconcerts and discotheques there is a large variation in the actual noise levels during exposures between and within concert halls and discotheques. Also, the number of exposures per year or the annual hours of exposure usually varies in the course of years. In a study about the relationship of hearing threshold levels and exposure to pop-music through headphones, a study population was selected which was – as assessed from a national inventory of popmusic habits in the Netherlands – assumed to be still without or with minor exposure to popmusic at popconcerts and discotheques. The study comprised over 400 subjects
aged 14 to 20 years and the exposure of each subject to popmusic through headphones (and during other activities) was assessed in detail. It was made plausible that the model given in ISO 1999 for occupational exposure also holds, albeit with a slight adaptation, for this type of exposure (Passchier-Vermeer, 1998). Whether the model also applies to the much more irregular exposures of teenagers to pop-music at pop-concerts, discotheques and dance halls, is unknown. Based on the results of epidemiological surveys on hearing threshold levels of a random sample of the general population or parts of the general population (e.g. 18 years old recruits), Passchier-Vermeer (1993) concluded that by far the largest part of the cumulative distribution of hearing threshold levels of the general population did not change in the last 25 years or so. Given the data of secondary school children in the Netherlands, this conclusion has been confirmed for the Dutch situation. However, the results, based on screening audiometry of two large populations of young people in Austria and Norway at the end of the eighties showed a serious deterioration of hearing of young males and females (17 – 18 years old), which was attributed to popmusic activities. This observation is not supported by Netherlands, Swedish and German investigations, based on threshold audiometry of smaller groups of young people. Most probably procedures related to mass screening techniques caused a systematic increase in hearing threshold levels in the Austrian and Norwegian populations.

5.3 Conclusion

Although noise-induced hearing impairment among teenagers has been reported in isolated cases, a comparison of the present distributions of hearing threshold levels of young populations with those distributions 30 years ago fails to show increases in this distribution.
6 References


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7 Figures

Figure 1 Physiological effects of noise on a pre-term baby

Figure 2 Susceptibility for hearing impairment. Figure 2a the results for mice, figure 2b for human beings.
Figure 3  Sleep wake stages for various ages. Percentage of the 24 hours spent in a given stage.

Figure 4  Effects of aircraft noise and sonic booms during slow wave sleep (SWS sleep) for children and adults (Lukas, 1972).
Figure 5  EEG arousals and awakenings of children as a result of noise exposure (Busby et al., 1985).
Appendix A  Characterisation of noise exposure

Sound pressure level and sound level
Sound is a physical phenomenon with alternating compression and expansion of air, which propagates from a source in all directions. These alternating compressions and expansions can be described as small changes in pressure around the atmospheric pressure. The frequency of the alternations determines the pitch of a sound: a high pitched tone (e.g. 4000 Hz) has a squeaking sound, a low pitched tone (e.g. 200 Hz) a humming sound. Sound pressures, relative to the atmospheric pressure, range from less than 20 micropascal up to more than 200 pascal, a range of 1 to 10 million. Therefore, in acoustics, the logarithm of the sound pressure relative to a reference sound pressure is taken as a basis for a sound exposure measure: the physical quantity sound pressure level expressed in decibel (dB).

The human hearing organ is not equally sensitive to sounds at different frequencies. Therefore, a spectral sensitivity factor is used which rates the sound pressure levels at the different frequencies in a comparable way as the adult human hearing organ does: the so-called A-weighting. The biophysical quantity A-weighted sound pressure level (symbol $L_{A}$) is expressed in dB(A) and is referred to as sound level.

Long term noise exposure

Equivalent sound level
The sound level is the basic metric from which other biophysical metrics to specify long-term exposure to noise are derived. In environmental and occupational situations the sound level fluctuates with time. From these fluctuating sound levels, the equivalent sound level (symbol $L_{eq,T}$) over a period of time $T$ is determined from:

$$L_{eq,T} = 10 \log_{10} \left( \frac{1}{T} \int_{0}^{T} \left( \frac{10}{10} \right)^{L(t)} \, dt \right)$$

In the equivalent sound level over a period $T$, the highest sound levels occurring during this period are counted more heavily than in the ‘normal’ average sound level over period $T$. This is demonstrated by the example given in figure A1. It shows the typical situation in an incubator. The average sound level over the period shown in the figure is 60 dB(A), the equivalent sound level over that period is 70 dB(A).

Common exposure periods $T$ to assess environmental or occupational noise exposure are 24 hours (full day) and 8 hours (working day).

For environmental health assessment purposes, usually a noise metric is assessed on an annual basis. In various countries, the so-called day-night level ($L_{dn}$) is in use. This metric is the equivalent sound level over 24 hours, with the sound levels during the night (period of 23:00 - 07:00 h) increased by 10 dB(A). Also a ‘day-evening-night level’ ($L_{den}$) is used, which is constructed similarly, be it that the sound levels during the evening (19:00-23:00 h) are increased by 5 dB(A),
and those during the night (23:00-07:00 h) by 10 dB(A). Commonly \( L_{dn} \) or \( L_{dnen} \) are measured in front of the facade of residential buildings.

![Graph of sound level over time](image)

**Figure A1 Characterisation of long term noise exposure.** As an example, the sound level (in dB(A)) is given as a function of time (in minutes). The average sound level over the time registered is 60 dB(A), the equivalent sound level over that time 70 dB(A).

**Single noise event**

**Specification of the noise of a single noise event**

In figure A2 the sound levels of an isolated noise event are given as a function of time. The noise comes from a single shot of a toy pistol and is measured at a distance of 20 cm from the toy in the direction of the barrel. The noise from such an event can be specified by its maximal level, sound exposure level, or peak sound pressure level.

**Maximal level**

To assess the so-called maximal level, several time-averaging networks of a noise level meter may be used, such as S (averaging time 1 s) and F (averaging time 125 ms).

**Sound exposure level**

If a noise event is of a short duration, less than one second, the sound exposure level or SEL of the event is equal to the equivalent sound level measured over 1 s. If the event is of a longer duration, the sound exposure level or SEL is the equivalent sound level during the event normalised to a period of one second.
**Peak sound pressure level**

A single noise event of very short duration, such as the noise impulse from a toy pistol, may also be specified by its peak sound pressure level. To assess this peak value, the measurement time is in the order of about 50 to 100 micro seconds. Usually no frequency weighting system is used if the peak sound pressure level is measured.

**The example**

In figure A2 the noise measures of the noise impulse accompanying a shot with a toy pistol are given. The peak sound pressure level is 150 dB at a distance of 20 cm (this is a typical value, see Passchier-Vermeer, 1991). Given a typical duration in the order of milliseconds, the maximal level on F is equal to 131 dB(A), SEL is 122 dB(A), \( L_{Aeq,1h} \) is 87 dB(A) and \( L_{Aeq,24h} \) 73 dB(A). For 100 of these pistol shots \( L_{Aeq,24h} \) is equal to \( 73 + 20 = 93 \) dB(A) \( (73 + 10 \times \log_{10} 100 = 93) \).

![Figure A2 Characterisation of a noise event. As an example the noise of a pistol when fired once is given. The sound level is given as a function of time (in millisecond).]
Appendix B  Effects of environmental noise on adults

Introduction

The effects of noise exposure on adults are:
• Hearing impairment
• Psycho-social effects
• Stress-related somatic health effects
• Effects on sleep
• Effects on cognitive performance

Noise-induced hearing impairment

Hearing impairment is an increase in hearing threshold level (ISO 1999, 1990). Prolonged noise exposure is able to destroy hair cells of the cochlea. This destruction of hair cells has the effect of a hearing impairment in the higher frequency range of the hearing threshold, especially in the range of 3000 to 6000 Hz, with largest effects at 4000 Hz. The effects of prolonged noise exposure and of ageing on hearing threshold levels are to a large extent additive. The results of various studies strongly suggest that the model presented in ISO 1999 for occupational noise exposure can also be accepted for environmental and leisure time noise exposures of adults and older children, provided the exposures are not too extreme and the exposures are expressed in $L_{Aeq,24h}$ instead of $L_{Aeq,8h}$. This implies that exposure to environmental and leisure time noise with $L_{Aeq,24h}$ values below 70 dB(A) does not cause hearing impairment in the large majority of adults (over 95%), even in case of life time exposure. It should be borne in mind, however, that there are no large-scale epidemiological studies that investigated noise-induced hearing impairment in the general population that do support this statement. Also, data from animal experiments indicate that young children may be more vulnerable in acquiring noise-induced hearing impairment than adults (Passchier-Vermeer, 1991). This will be discussed in more detail in section 4.2.

For impulsive (shooting) noise with $L_{Aeq,24h}$ over 80 dB(A), studies on temporary threshold shifts suggest the possibility of an increased risk for impulse noise-induced hearing impairment in adults (Smoorenburg, 1998).

Psycho-social effects

The main psycho-social effect is annoyance. Noise annoyance is a feeling of resentment, displeasure, discomfort, dissatisfaction or offence when noise interferes with someone’s thoughts, feelings or actual activities. Noise annoyance in populations is evaluated by using questionnaires. Exposure-effect relationships for adult populations have been derived for exposure to the three main types of traffic noise: road, and railway traffic and aircraft (Miedema et al., 1998). Environmental noise exposure is only one of the factors that contribute to noise annoyance, albeit a significant one (Job, 1996; Job, 1999; Miedema et al., 1999; Guski, 1999; Stallen, 1999). The degree of annoyance experienced by an individual, but also on a population level can in practice differ considerably from the general exposure-response relationships, because of the influence of non-acoustical effect-modifying factors. Such factors are anxiety, fear of the noise source and the
feeling that the noise could be avoided. Other possible noise-induced psycho-social effects, that have been studied in adult populations, are: social isolation, aggression, and depression. It has also been studied whether in areas with high environmental noise exposure admission to mental hospitals is larger than in quiet surroundings.

**Stress-related somatic health effects**

A large number of laboratory experiments have shown noise-induced acute temporal changes in many physiological systems, including the cardiovascular system. These findings led to several investigations into possible long-term effects associated with noise exposure, such as stress-related cardiovascular disorders (Babisch et al., 1998). In addition some research has been carried out regarding effects on the hormone and immune system, that are also related to stress-induced somatic changes. There is sufficient evidence that high environmental noise exposures increase the risk for ischaemic heart disease and hypertension in adult populations. According to stress models neuroendocrine reactions act as mediator in the organism on the pathway from noise exposure, via emotional and cognitive processes to physiological and biochemical changes in the human body, which are associated with an increased risk of diseases. Therefore, although not being a risk factor as such, stress hormones like epinephrine, norepinephrine and cortisol are indicators of the arousal of the sympathetic-adrenal system. There is some evidence from epidemiological surveys that epinephrine and norepinephrine levels are higher in adult populations exposed to high levels of environmental noise.

**Effects on sleep**

Sleep is a recovery process that is essential for humans to function properly and in the extreme necessary to survive. Sleep is as essential for human life as water. Besides, people like to sleep and usually consider a good night’s sleep to be an important aspect of an individual’s quality of life. Adverse health effects are expected from chronic noise-induced interference with sleep, as it impairs the functions of sleep with respect to brain and body restoration (Horne, 1990; Carter, 1998). Apart from the physiological aspects of a noise-induced reduction of sleep quality, night-time noise exposure of sufficient intensity is also related to subjectively experienced sleep quality (Passchier-Vermeer et al., 1998). Also, reduced sleep quality interferes with daytime functioning, having an adverse effect on mood of adults next day and possibly also on cognitive performance of adults.

Sleep quality can be quantified by subjective and objective methods. The most commonly applied subjective methods are self-reporting using sleep logs or diaries. The most commonly used objective methods are EEG recordings and actimetry. From the sleep EEG, usually considered the golden standard for sleep research, two distinct phases of sleep are distinguished. These phases are: NREM sleep and REM sleep, also called dream sleep (REM is rapid eye movement). NREM sleep covers four stages, stages 3 and 4 are called deep sleep (slow wave sleep: SWS) and stages 1 and 2 light sleep (in these stages the transition from SWS sleep to REM sleep or awakening occurs). During NREM sleep mainly body restoration occurs and during REM sleep brain restoration. Recently it was shown that memory consolidation, as part of brain restoration, not only takes place during REM sleep, but that SWS in the first part of the night contributes signifi-
cantly to memory consolidation (Stickgold, 1998). There are essential physiological differences between NREM and REM sleep. With respect to the central nervous system: during NREM sleep the parasympathetic tone increased and the sympathetic tone is reduced, especially during SWS sleep. In REM sleep sympathetic activity is highly variable, which results in e.g. changes in blood pressure of 10 to 40 mm Hg and increased irregularity in breathing. During NREM there is a decrease in body temperature, and during REM the thermoregulatory responses are attenuated. Growth hormone is mostly released during SWS sleep. With respect to the immune system: Interleukin-1 levels increase during sleep, and peak levels occur at the onset of SWS. With respect to cerebral blood flow and metabolism: there is a reduction of blood flow by 25 to 45% in SWS sleep compared to values while awake, and in REM values are about equal to values while awake.

During sleep, adults have sleep cycles of about 90 minutes in which REM and NREM sleep occur alternating (see figure B1). Adults spend about 80% of the sleeping period in NREM (25% in SWS, 50% in stage 2, 5% in stage 1) and 20% in REM. In figure 3 of the main text the time spent in the various wake sleep stages are given for human beings of various ages. For one week-old babies the average sleep length is 16 hours (with a standard deviation of 2 hr). After a year the average sleep length is 13 hours. Total sleep time declines almost entirely as a result of the decline of REM sleep from 8 hours at birth to 4 hours at one year. After about one year the sleep pattern of babies is about that of adults, be it that the cycle REM NREM is 60 to 70 minutes. Regular day-time naps disappear by about 5 years. Total amount of sleep decreases to 12 hours at 2 years, 10 hours at 5 years, 9 hours at six to seven years, 8.5 hours at 8-9 years, 8 hours at 10-11 years. The sleep cycle is 80 to 90 minutes for the average 5 years old, REM sleep has then reached the adult proportion of 20 to 25%. SWS starts to occur at about 4 months, is about 50% of NREM at 1 year, and remains at this high level for several years. By age of 9 years SWS sleep is 22 to 28% of sleep time.

In the eighties a series of epidemiological and laboratory sleep studies have been carried out in the framework of a research program of the European Commission. From a joint analysis of the EEG results it could be shown only that night-time noise exposure caused a decrease of time spent in REM of 7 minutes and no other statistically significant changes could be established (Jurriëns, 1983). Several epidemiological studies and synopses have produced exposure effect relationships between the probability of awakening due to a noise event and a noise measure of the event (Fidell et al., 1995; Fidell et al., 1998; Ollerhead et al., 1992; Passchier-Vermeer, 1994; Passchier-Vermeer et al., 1998; Pearsons, 1996; Pearsons et al., 1989).
Figure B1 Sleep pattern of a young adult. Distribution of W (waking), REM sleep and NREM sleep consisting of stage 1, 2, 3, and 4. Stages 3 and 4 of NREM are also called slow wave sleep (SWS sleep).

Effects on performance
From laboratory experiments there is overwhelming evidence that the presence of uncontrollable noise can significantly impair cognitive performance of adults (Cohen, 1980; Cohen et al., 1977; Cohen et al., 1986). Noise is able to induce learned helplessness, increase arousal, alter the choice of task strategy, and decrease attention to the task (Smith, 1990). Noise may also affect social performance, mask speech and other relevant sound signals, impair communication and it may distract attention from relevant social clues. Already at low levels adverse acute effects have been assessed. Performance on a task involving motor and monotonous activities is sometimes not decreased, but on the contrary enhanced. Epidemiological studies on the effect of environmental noise on adult performance are lacking.