NATIONAL SIDS/INFANT DEATH RESOURCE CENTER

Publications added to NSIDRC collection for October 2004

Accession Number	Title

PA-01143

Getting through the holidays when you've lost a loved one.

Sims DD.
St. Meinrad, IN : Abbey Press. 2004, 4 p.

This booklet discusses how the holidays can be a painful time after losing a loved one, provides tips that can make it a time to cherish fond memories and traditions. Author guides through the holidays and beyond so that focus will be on loved one's life not their death. He provides ideas that may help begin the journey like being patient and realistic, listening to heart and acknowledging limits, adapting cherished traditions, allowing tears to come, but looking for joy amidst the pain, focusing on the spiritual dimension of the holidays. Sources of additional help are identified like books magazine on bereavement. Refer to PA-01144 for spanish language version of the booklet.

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PA-01144

Sobreviviendo a los dias festivos cuando has perdido a un ser amando [Getting through the holidays when you've lost a loved one].

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Accession Number	Title

PA-01145

Losing a Child : Explorations in Grief.

Hurcombe L
London, UK : Sheldon Press. 200 , 179 p.

This book gives an understanding of how grieving for a child affects every member of the family, and the relationships between the surviving members. Explains the different types of support available and how they can be accessed. A special chapter deals with sudden or violent death: murder, accidents and suicide. The main focus is on losing children aged 0-18. Includes helpful contacts, references, reading list and index.

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PA-01146

Hope is like the Sun : Finding hope and healing after miscarriage, stillbirth, or infant death.

Church, L.
Hampton, VA : HopeXchange Publishing. 2004, 174 p.

This book offers support to women and their families coping with pregnancy loss. The author and four of her friends narrate their own experience with miscarriage and offer advice and supportive information to bereaved parents and those who care for them. Through personal narratives, author illustrates the many facets of the normal grieving process that accompany pregnancy loss. Each chapter presents user-friendly suggestions for coping with a wide range of emotional responses, assuring women that reactions are normal and not crazy. Includes information on self-care, importance of maintaining physical health while healing emotionally, suggestions to overcome social and spiritual pain of grief, ways to commemorate the loss of a baby, handling holidays and special events, dealing with family members and other siblings, handling spouses grief, tips to aid friends and loved ones of grieving parents, decision to have another baby. Appendices include resources such as books, websites, national organizations, support groups contact information, glossary and index. Each chapter includes workbook type sections.

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PA-01147

21st century complete medical guide to Sudden Infant Death Syndrome (SIDS): authoritative government documents, clinical references, and practical information for patients and physicians (CD-ROM).

Progressive Management Medical Health News

Accession Number	Title

: . 2004, 32742 p.

This up-to-date electronic book on CD-ROM provides collection of official Federal government information and documents on SIDS. It presents clinical and medical information from authoritative sources like National Institutes for Health (NIH), Food and Drug Administration (FDA), Combined Health Information Database (CHID), Centers for Disease Control (CDC), and the National Institute of Child Health and Human Development (NICHD). It includes an encyclopedic collection of general medical and health documents, thousands of pages with extensive material from the CDC and NIH on hundreds of diseases and health topics from A to Z, along with FDA drug and medical publications, government consumer healthcare tips, disease prevention programs, dietary guidelines, and travelers' health information. It provides exclusive "Guide to Leading Medical Websites" with updated links to 73 of the best sites for medical information. This CD-ROM has over 32,000 pages reproduced using Adobe Acrobat PDF software and Reader software is included.

Distributed by:

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http://www.directtextbook.com; http://www.amazon.com

SIDS-07017 **Autonomic responses to sighs in healthy infants and in victims of sudden infant death.**

Franco P, Verheulpen, D et al.
Sleep Medicine , 4 (6): 569-77, 2003

Objective: Sigh, defined as an isolated breath with an increased tidal volume, can be associated with abrupt changes in heart rate (HR) or blood oxygenation. Sigh may be followed by a central apnea. As impairment of autonomic control was postulated in future SIDS victims, we hypothesized that their autonomic responses to sighs were different from those of healthy control infants. Methods: Sighs followed by central apnea were studied in the sleep recordings of 18 infants who eventually died of SIDS and of 18 control infants. The infants of the two groups were matched for sex, gestational age, postnatal age, weight at birth and sleep position during sleep recording. HR autoregressive power spectral analysis was performed on RR intervals preceding and following sighs. Results: In all infants, most sighs followed by an apnea were found in NREM sleep. Compared to the control infants, the future SIDS victims were characterized by a greater sympathovagal balance and a lower parasympathetic tonus before the sighs. Following the sighs, no more differences were found in NREM sleep. Conclusion: Based on the present findings, it can be postulated that sighs contribute to reset autonomic tonus during NREM sleep.

SIDS-07018 **The Sudden infant death syndrome gene: Does it exit?**

Opdal SH, Rognum TO.
Pediatrics , 114 (4): e506-12, Oct 2004
For Full text: http://www.pediatrics.org

Accession Number	Title

Background: Sudden infant death syndrome (SIDS) is in a difficult position between the legal and medical systems. In the United Kingdom, prosecutors have for years applied the simple rule that 1 unexpected death in a family is a tragedy, 2 are suspicious, and 3 are murder. However, it seems that the pendulum has now swung to the opposite extreme; mutations or polymorphisms with unclear biological significance are accepted in court as possible causes of death. This development makes research on genetic predisposing factors for SIDS increasingly important, from the standpoint of the legal protection of infants. The genetic component of sudden infant death can be divided into 2 categories, ie (1) mutations that give rise to genetic disorders that constitute the cause of death by themselves and (2) polymorphisms that might predispose infants to death in critical situations. Distinguishing between these 2 categories is essential, and cases in which a mutation causing a lethal genetic disorder is identified should be diagnosed not as SIDS but as explained death. Genetic alterations that may cause Sudden Infant Death. Deficiencies in fatty acid metabolism have been extensively studied in cases of SIDS, and by far the most well-investigated mutation is the A985G mutation in the medium-chain acyl-CoA dehydrogenase (MCAD) gene, which is the most prevalent mutation causing MCAD deficiency. However, <1% of sudden infant death cases investigated have this mutation, and findings of biochemical profiles seen in specific fatty acid oxidation disorders in a number of such cases emphasize the importance of investigating fatty acid oxidation disorders other than MCAD deficiency. Severe acute hypoglycemia may cause sudden death among infants, but only rare novel polymorphisms have been found when key proteins involved in the regulation of blood glucose levels are investigated in cases of SIDS. The long QT syndrome (LQTS) is another inherited condition proposed as the cause of death in some cases of sudden infant death. The LQTS is caused by mutations in genes encoding cardiac ion channels, and mutations in the genes KVLQT1 and SCNA5 have been identified in cases initially diagnosed as SIDS, in addition to several polymorphisms in these 2 genes and in the HERG gene. In addition, genetic risk factors for thrombosis were investigated in a small number of SIDS cases; the study concluded that venous thrombosis is not a major cause of sudden infant death. Gene Polymorphisms that may predispose infants to Sudden Infant Death under certain circumstances. Many SIDS victims have an activated immune system, which may indicate that they are vulnerable to simple infections. One reason for such vulnerability may be partial deletions of the complement component 4 gene. In cases of SIDS, an association between slight infections before death and partial deletions of the complement component 4 gene has been identified, which may indicate that this combination represents increased risk of sudden infant death. There have been a few studies investigating HLA-DR genotypes and SIDS, but no association has been demonstrated. The most common polymorphisms in the interleukin-10 (IL-10) gene promoter have been investigated in SIDS cases, and the ATA/ATA genotype has been reported to be associated with both SIDS and infectious death. The findings may indicate that, in a given situation, an infant with an unfavorable IL-10 genotype may exhibit aberrant IL-10 production, and they confirm the assumption that genes involved in the immune system are of importance with respect to sudden unexpected infant death. Another gene that has been investigated is the serotonin transporter gene, and an association between the long alleles of this gene and SIDS has been demonstrated. Serotonin influences a broad range of physiologic systems, as well as the interactions between the immune and nervous systems, and findings of decreased serotonergic binding in parts of the brainstem, together with the findings in the serotonin transporter gene, may indicate that serotonin plays a regulatory role in SIDS. It has also been speculated that inadequate thermal regulation is involved in SIDS, but investigations of genes encoding heat-shock proteins and genes encoding proteins involved in lipolysis from brown adipose tissue have not found evidence of linkages between common polymorphisms in these genes and SIDS. A number of human diseases are attributable to mutations in mitochondrial DNA (mtDNA), and there are several reasons to think that mtDNA mutations also are involved in SIDS. Both a higher substitution frequency and a different substitution pattern in the HVR-I region of mtDNA have been reported in SIDS cases, compared with control cases. A number of coding region mtDNA mutations have also been reported, but many are found only in 1 or a few SIDS cases, and, to date, no predominant mtDNA mutation has been found to be associated with SIDS. Conclusions: All mutations giving rise to metabolic disorders known to be associated with life-threatening events are possible candidates for genes involved in cases of sudden infant death, either as a cause of death or as a predisposing factor. It is necessary to distinguish between lethal mutations leading to diseases such as MCAD and LQTS, and polymorphisms (for instance, in the IL-10 gene and mtDNA) that are normal gene variants but might be suboptimal in critical situations and thus predispose infants to sudden infant death. It is unlikely that one mutation or polymorphism is the predisposing factor in all SIDS cases. However, it is likely that there are "SIDS genes" operating as a polygenic inheritance predisposing infants to sudden infant death, in combination with environmental risk factors. For genetically predisposed infants, a combination of, for instance, a slight infection, a prone sleeping position, and a warm environment may trigger a vicious circle with a death mechanism, including hyperthermia, irregular breathing, hypoxemia, and defective autoresuscitation,

Accession Number	Title

SIDS-07019

Sudden unexpected death in infancy: A multi-agency protocol for care and investigation.

The Royal College of Pathologists and the Royal College of Paediatrics and Child Health
London, United Kingdom : The Royal College of Pathologists and the Royal College of Paediatrics and Child Health , 2004, 73 p.

This protocol is by a working group of The Royal College of Pathologists and The Royal College of Paediatrics and Child Health. All agencies involved in sudden deaths in infancy have collaborated to recommend how these tragic events should be investigated. The aims are, first, to prevent miscarriages of justice and the unwarranted prosecution of parents and, second, to protect the welfare and safety of infants and children.
The Working Group's extensive recommendations include:
- Introduction of a compulsory national investigation protocol
- In court cases where expert testimony is central, a pre-trial meeting of experts should establish areas of disagreement and set them out in writing for the court
- Better training of all personnel involved in investigating sudden death in infancy
- Inquests should be held in all cases of sudden death in infancy, except where there are immediately recognisable natural causes, e.g. a congenital heart defect
- Decisions to prosecute should be made only after reference to a multi-professional review.
Includes references and Appendices 1-6.

Distributed by:
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SIDS-07020

Smoking during pregnancy- United States- 1990-2002.

Mathews TJ, Rivera CC,
Morbidity and Mortality Weekly Report , 53 (39): 911-5, Oct 8, 2004
For Full text: http://www.cdc.gov/mmwr

Cigarette smoking during pregnancy adversely affects the health of both mother and child. The risk for adverse maternal conditions (e.g., premature rupture of membranes, abruptio placentae, and placenta previa) and poor pregnancy outcomes (e.g., neonatal mortality and stillbirth, preterm delivery, and sudden infant death syndrome) is increased by maternal smoking. Infants born to mothers who smoke weigh less than other infants, and low birthweight (<2,500 grams) is a key predictor for infant mortality. Infertility and conception delay also might be elevated by smoking. National health objectives for 2010 target an increase in cessation to 30% among pregnant smokers during the first trimester and abstinence from cigarettes by 99% of women giving birth. To assess progress toward these goals, CDC analyzed state-specific trends in maternal smoking during 1990-2002 by using data collected on birth certificates. This report summarizes the results of those analyses, which indicated that whereas participating areas observed a significant decline in maternal smoking during the surveillance period, 10 states reported recent increases in smoking by pregnant teens. Although the widespread public health message to abstain from smoking during pregnancy has helped decrease maternal smoking, to reduce prevalence further, implementation of additional interventions are required.

Accession Number	Title

SIDS-07021

Influence of lung fixation technique on the state of alveolar expansion - a histomorphometrical study.

Hausmann R, Bock H, Biermann T, Betz P.
Leg Med (Tokyo) , 6 (1): 61-5, Mar 2004
For Full text: http://www.sciencedirect.com

Lungs removed at necropsy normally collapse due to the loss of negative pleural pressure leading to a quite unnatural appearance of both gross and histological specimens. In order to demonstrate the influence of post-mortem lung retraction on the degree of alveolar expansion, a histomorphometrical analysis was performed in lungs from a 9-month-old healthy infant. Tissue specimens from the right lung were obtained at autopsy and routinely fixed after retraction ('routinely fixed lung'), whereas the left lung was fixed in situ before opening the thoracic cage ('in situ fixed lung'). The size of the alveoli as well as the thickness of the alveolar walls were measured using an automatic image processing and analysis system (Leica QWIN) in both lungs. The mean alveolar size was 8.7 x 10(3) microm(2) in the routinely fixed lung (alveoli, n=1.1576) and 10.9 x 10(3) microm(2) in the in situ fixed lung (alveoli, n = 841). In contrast, the diameter of the alveolar walls showed no significant difference in both lungs. The average thickness of the alveolar walls was 7.9 microm (measuring sites, n = 1.190) in the routinely fixed lung and 8.1 microm in the in situ fixed lung (measuring sites, n = 1.027), respectively. The results provide evidence of significantly reduced aeration in the retracted and routinely fixed lung which could be of special forensic interest in cases of suspicious infanticide, stillbirth or infant death by drowning or suffocation.

SIDS-07022

Endogenous 5-HT(1/2) systems and the newborn rat respiratory control. A comparative in vivo and in vitro study.

Bodineau L, Cayetanot F et al.
Respir Physiol Neurobiol , 141 (1): 47-57, Jul 2004
For Full text: http://www.sciencedirect.com

Consequences of 5-HT(1/2) systems blockade by methysergide on newborn rats respiratory drive were evaluated in vivo with unrestrained animals and in vitro using brainstem-spinal cord preparations. A decrease in respiratory frequency until a plateau level was observed under both in vivo (82.8 +/- 0.6% of control values) and in vitro (76.8 +/- 0.8% of control values) conditions whereas an increase in inspiratory amplitude (135.1 +/- 2.1% of control values) was only retrieved in vivo. By the use of the c-fos expression analysis, we correlated these effects with neuronal activity changes, particularly, in vivo in two key structures between the respiratory ponto-medullary network and the peripheral or suprapontine afferences, namely the commissural subnucleus of the nucleus of the solitary tract and the lateral parabrachial nucleus. Thus, peripheral and suprapontine inputs seem to be of a primeval importance in the respiratory influence of endogenous 5-HT. Besides, as 5-HT is involved in the respiratory perturbations that occur in sudden infant death syndrome (SIDS), our results suggest a participation of peripheral and suprapontine inputs in these disorders.

SIDS-07023

Does particulate air pollution contribute to infant death? A systematic review.

Glinianaia SV, Rankin J, Bell R, Pless-Mulloli T, Howel D.
Environ Health Perspect , 112 (14): 1365-71, Oct 2004

Accession Number	Title

There is now substantial evidence that both short- and long-term increases in ambient air pollution are associated with increased mortality and morbidity in adults and children. Children's health is particularly vulnerable to environmental pollution, and infant mortality is still a major contributor to childhood mortality. In this systematic review we summarize and evaluate the current level of epidemiologic evidence of an association between particulate air pollution and infant mortality. We identified relevant publications using database searches with a comprehensive list of search terms and other established search methods. We included articles in the review according to specified inclusion criteria. Fifteen studies met our inclusion criteria. Evidence of an association between particulate air pollution and infant mortality in general was inconsistent, being reported from locations with largely comparable pollution levels. There was some evidence that the strength of association with particulate matter differed by subgroups of infant mortality. It was more consistent for postneonatal mortality due to respiratory causes and sudden infant death syndrome. Differential findings for various mortality subgroups within studies suggest a stronger association of particulate air pollution with some causes of infant death. Research is needed to confirm and clarify these links, using the most appropriate methodologies for exposure assessment and control of confounders.

SIDS-07024

Unexpected infant death: Lessons from the Sally Clark case.

Byard RW.
Med J Aust , 181 (1): 52-4, Jul 2004
For Full text: http://www.mja.com.au

In November 1999, in the United Kingdom, a woman was convicted of the murder of her two infant sons. An appeal against the conviction was dismissed in October 2000, but the conviction was quashed by a second court of appeal in January 2003. Review of the autopsy findings showed that standard procedures had not always been followed, thus limiting verification of the alleged findings. Some potentially important diagnoses and conclusions were also altered over time. This case and its sequelae demonstrate the difficulties that may arise if cases are not fully investigated by pathologists with specific training or experience in paediatric forensic pathology, with all of the results being clearly summarised and discussed in autopsy reports. Trying to clarify findings, diagnoses and circumstances of death at a later stage may simply not be feasible, owing to a wide variety of possibilities other than inflicted injury. This type of case has unfortunately led to mistrust of the medical and legal systems and has made the investigation of such emotive and tragic cases all the harder.

SIDS-07025

Prenatal nicotine exposure alters the types of nicotinic receptors that facilitate excitatory inputs to cardiac vagal neurons.

Huang ZG, Wang X, Evans C, Gold A, Bouairi E, Mendelowitz D.
J Neurophysiol , 92 (4): 2548-54, Jun 22 2004
For Full text: http://jn.physiology.org/

Accession Number	Title

Nicotinic receptors play an important role in modulating the activity of parasympathetic cardiac vagal neurons in the medulla. Previous work has shown nicotine acts via at least three mechanisms to excite brain stem premotor cardiac vagal neurons. Nicotine evokes a direct increase in holding current and facilitates both the frequency and amplitude of glutamatergic neurotransmission to cardiac vagal neurons. This study tests whether these nicotinic receptor-mediated responses are endogenously active, whether alpha4beta2 and alpha7 nicotinic receptors are involved, and whether prenatal exposure to nicotine alters the magnitude of these responses and the types of nicotinic receptors involved. Application of neostigmine (10 microM) significantly increased the holding current, amplitude, and frequency of miniature excitatory postsynaptic current (mEPSC) glutamatergic events in cardiac vagal neurons. In unexposed animals, the nicotine-evoked facilitation of mEPSC frequency, but not mEPSC amplitude or holding current, was blocked by alpha-bungarotoxin (100 nM). Prenatal nicotine exposure significantly exaggerated and altered the types of nicotinic receptors involved in these responses. In prenatal nicotine-exposed animals, alpha-bungarotoxin only partially reduced the increase in mEPSC frequency. In addition, in prenatal nicotine-exposed animals, the increase in holding current was partially dependent on alpha-7 subunit-containing nicotinic receptors, in contrast to unexposed animals in which alpha-bungarotoxin had no effect. These results indicate prenatal nicotine exposure, one of the highest risk factors for sudden infant death syndrome (SIDS), exaggerates the responses and changes the types of nicotinic receptors involved in exciting premotor cardiac vagal neurons. These alterations could be responsible for the pronounced bradycardia that occurs during apnea in SIDS victims.

SIDS-07026

Sudden infant death syndrome due to parainfluenza virus 2 associated with hemophagocytic syndrome.

Kashiwagi Y, Kawashima H, et al.
J Infect , 49 (4): 329-32, Nov 2004
For Full text: http://www.sciencedirect.com

We report a child with Sudden Infant Death Syndrome (SIDS), aged 16 months. The histological findings of tonsils, spleen, and bone marrow revealed many hemophagocytic cells. Parainfluenza virus type 2 (PIV2) was cultured in the nasopharynx and detected by reverse-transcription (RT)-PCR in liver tissue and bone marrow. His laboratory data of elevated level of ferritin and IL-6 suggested hemophagocytic syndrome (HPS). It is suspected that PIV2 infection in infants is a risk factor for SIDS.

SIDS-07027

Seasonal variation of sudden infant death syndrome in Hawaii.

Mage DT.
J Epidemiol Community Health , 58 (11): 912-16, Nov 2004
For Full text: http://jech.bmjjournals.com

Accession Number	Title

Objective: To test whether the sudden infant death syndrome (SIDS) rate displays the universal winter maximum and summer minimum in Hawaii where there is no appreciable seasonal variation of temperature. Design: The null hypothesis is tested that there is no seasonal variation of necropsied SIDS in Hawaii. The numbers of live births and SIDS cases by month for the years 1979 to 2002 were collected and the monthly SIDS distribution is predicted based on the age at death distribution. Setting: The state of Hawaii, located in the midst of the Pacific Ocean, has a semi-tropical climate with temperatures fluctuating diurnally as 25 +/- 5 degrees C throughout the year. Therefore homes are unheated and infants are not excessively swaddled. The Hawaii State Department of Health maintains vital statistics of all infant births and deaths. Main Results: The results reject the null hypothesis of no seasonal variation of SIDS ($p = 0.026$). An explanation for the seasonal effect of the winter maximum and summer minimum for Hawaiian SIDS is that it arises from the cycle of the school session and summer vacation periods that represent variable intensity of a possible viral infection vector. SIDS rates in both Hawaii and the United States increase with parity, also indicating a possible role of school age siblings as carriers. Conclusions: The winter peak of the SIDS in Hawaii is support for the hypothesis that a low grade viral infection, insufficient by itself to be a visible cause of death at necropsy, may be implicated as contributing to SIDS in vulnerable infants.

SIDS-07028

Phenotypic manifestations of the OCTN2 V295X mutation: Sudden infant death and carnitine-responsive cardiomyopathy in Roma families.

Melegh B, Bene J, Mogyorosy G, et al.
Am J Med Genet , ( ): [e-pub ahead of print], Oct 2004
For Full text: http://www3.interscience.wiley.com

In two non-consanguineous Hungarian Roma (Gypsy) children who presented with cardiomyopathy and decreased plasma carnitine levels, we identified homozygous deletion of 17081C of the SLC22A5 gene that results in a frameshift at R282D and leads ultimately to a premature stop codon (V295X) in the OCTN2 carnitine transporter. Carnitine treatment resulted in dramatic improvement of the cardiac symptoms, echocardiographic, and EKG findings in both cases. Family investigations revealed four sudden deaths, two of them corresponded to the classic SIDS phenotype. In postmortem tissue specimens available from three of them we could verify the homozygous mutation. In liver tissue reserved from two patients lipid droplet vacuolization could be observed; the lipid vacuoles were located mainly in the peripherolobular regions of the acini. In the heart tissue signs of generalized hypertrophy and lipid vacuoles were seen predominantly in the subendocardial areas in both cases; some aggregates of smaller lipid vacuoles were separated, apparently by membranes. Review of all OCTN2 deficiency cases reported so far revealed that this is the first presentation of histopathology in classic familial sudden infant death syndrome (SIDS) with an established SLC22A5 mutation. In addition to the two affected homozygous cardiomyopathic children and three homozygous sudden death patients, the genetic analysis in 25 relatives showed 14 carriers. The mutant gene derived from five non-consanguineous grandparents, each of them having 6-14 brothers and sisters. This alone suggests a wide ancestral spread of the mutation in certain Roma subpopulations.

SIDS-07029

Frequency of bed sharing and its relationship to breastfeeding.

McCoy RC, Hunt CE, Lesko SM, et al.
J Dev Behav Pediatr , 25 (3): 141-9, Jun 2004
For Full text: http://www.jrnldbp.com

Accession Number	Title

Bed sharing has been promoted as facilitating breastfeeding but also may increase risks for sudden, unexpected infant deaths. This prospective cohort study was performed to determine the prevalence of adult and infant bed sharing and its association with maternal and infant characteristics. Demographic data were collected from 10,355 infant-mother pairs at birth hospitals in Eastern Massachusetts and Northwest Ohio, and follow-up data were collected at 1, 3, and 6 months by questionnaire. Associations with bed sharing were estimated using odds ratios and 95% confidence intervals from multiple logistic regression models while adjusting for confounding variables. At 1, 3, and 6 months, 22%, 14%, and 13% of infant-mother pairs shared a bed, respectively. On multivariate analysis, race/ethnicity and breastfeeding seemed to have the strongest association with bed sharing. These factors need to be considered in any comprehensive risk to benefit analysis of bed sharing.

SIDS-07030

Deaths : Final Data for 2002

Kenneth D, Kochanek MA, Sherry L, et al.
National Vital Statistics Reports , 53 (5): 1-116, Oct 12, 2004
For Full text: http://www.cdc.gov/nchs

Objectives: This report presents final 2002 data on US deaths and death rates according to demographic and medical characteristics such as age, sex, hispanic prigin, race, martial status, educational attainment, injury at work, state of residence, and cause of death. Trends and patterns in general mortality, life expectancy, and infant and maternal mortality are also described. A previous report presented preliminary mortality for 2002. Methods: In 2002 a total of 2,443,387 deaths were reported in the United States. This report presents descriptive tabulations of information reported on the death certificates. Funeral directors attending physicians, medical examiners and coroners complete death certificates. Original records are filed in the state registration offices. Statistical information is compiled into a national database through the Vital Statistics Cooperative Program of the National Center for Health Statistics (NCHS, Centers for Disease Control and Prevention, Causes of death are processed in accordance with the International Classification of Diseases Tenth Revision (ICD-10).Results: The age adjusted death rate for the United States in 2002 was 845.3 deaths per 100,00 standard population, representing a decrease of 1.1 percent from the 2001 rate and a record low historical figure. Life expectancy at birth rose by 0.1 year to a record high of 77.3 years. Considering all deaths, age-specific death rates rose only for those under 1 year and declined for a number of age groups including those 1-4 years, 25-34 years, 55-64 years, 65-74 years, 75-84 years, and 85 years and over. The 15 leading causes of death in 2002 remained the same as in 2001. Heart disease and cancer continued to be a leading and second leading causes of death, togrther accounting for over half of all deaths. The infant mortality rate in 2002 increaed to 7.0 compared with a rate of 6.8 in 2001. Conclusions: Generaly, mortality patterns in 2002 were consitent with long-term trends. Life expectancy in 2002 increased again to a new record level. The age-adjusted death rate declined to a record low historical figure. The age-adjusted death rate declined toa record low historical figure. However the infant mortality rate increaed in 2002. The infant mortality rate has either decreased or remained level each successive year through 2001 since 1958. Trends for homicide and injury at work wee interrupted due to the terrorist deaths that occurred September 11, 2001. The homicide rate decreased significantly from 2001 to 2002 and dropped to the 14th leading cause of death. Deaths due to injury at work also declined significantly during this period.

SIDS-07031

Prenatal nicotine exposure alters central cardiorespiratory response to hypoxia in rats: Implications for Sudden Infant Death Syndrome.

Neff RA, Simmens SJ, et al.
J Neurosci , 24 (42): 9261-8, Oct 20 2004
For Full text: http://www.jneurosci.org/

Accession Number	Title

Maternal cigarette smoking and prenatal nicotine exposure are the highest risk factors for sudden infant death syndrome (SIDS). During hypoxia, respiratory frequency and heart rate transiently increase and subsequently decrease. These biphasic cardiorespiratory responses normally serve to prolong survival during hypoxia by reducing the metabolic demands of cardiac and respiratory muscles. However, exaggerated responses to hypoxia may be life threatening and have been implicated in SIDS. Heart rate is primarily determined by the activity of brainstem preganglionic cardioinhibitory vagal neurons (CVNs) in the nucleus ambiguus. We developed an in vitro rat brainstem slice preparation that maintains rhythmic inspiratory-related activity and contains fluorescently labeled CVNs. Synaptic inputs to CVNs were examined using patch-clamp electrophysiological techniques. Hypoxia evoked a biphasic change in the frequency of both GABAergic and glycinergic IPSCs in CVNs, comprised of an initial increase followed by a decrease in IPSC frequency. Prenatal exposure to nicotine changed the GABAergic response to hypoxia from a biphasic response to a precipitous decrease in spontaneous GABAergic IPSC frequency. This study establishes a likely neurochemical mechanism for the heart rate response to hypoxia and a link between prenatal nicotine exposure and an exaggerated bradycardia during hypoxia that may contribute to SIDS.

SIDS-07032 **Ontogeny of arousal.**

Crowell DH, Brooks LJ, Corwin M, et al.
J Clin Neurophysiol , 21 (4): 290-300, Jul 2004
For Full text: http://www.clinicalneurophys.com

Ontogeny of arousal data constitute a vital supplement to the sparse literature on spontaneous neuronal activity. These data demonstrate that measurable infant spontaneous arousals (SAs) with an inherent oscillatory entrainment occur six times more in active sleep than in quiet sleep of the same duration and are identifiable as a human neurobiologic function. These SAs are not significantly associated with race or ethnicity, gender, total hours spent sleeping, percent time spent in active or quiet sleep, preterm status, history of a life-threatening event, having had a sibling who died of sudden infant death syndrome (SIDS), or having had a mother who smoked during this pregnancy. As measurable neurophysiologic events, SAs establish parameters for research at molecular and molar levels focusing on several critical areas: (1) the neuronal control of SA related to neurotransmitters, (2) as a significant antecedent factor in clinical cardiorespiratory events occurring in infants at high epidemiologic risk for SIDS; (3) as a regulatory biologic factor underlying temperament and executive cognitive functioning, and (4) morbidity and mortality effects possibly related to therapeutic interventions that alter SA levels.

SIDS-07033 **Parental smoking and passive smoking in infants: Fathers matter too.**

Blackburn CM, Bonas S, Spencer NJ, et al.
Health Educ Res , ( ): [e-pub], Aug 2004
For Full text: http://her.oupjournals.org/

Accession Number	Title

This study examines mothers' and fathers' smoking patterns in different kinds of smoking households, and assesses their relative contribution to infants' exposure to environmental tobacco smoke. It uses data from a cross-sectional survey of 314 smoking households (infants: mean age 10 weeks) in Coventry and Birmingham, England, examining reported tobacco consumption and objective measures of exposure: the study infant's urinary cotinine:creatinine ratios and their mother's salivary cotinine. The study shows that both mothers' and fathers' tobacco smoke make substantial contributions to infant exposure to tobacco smoke. Households were more likely to contain a smoking father than mother, with over two-thirds of households including a smoking father. In households where both parents smoke, fathers' tobacco consumption was found to be significantly higher than in households where only the father smokes. This suggests that the interaction between parents needs to be considered rather than focusing on mothers' or fathers' smoking behaviour in isolation. The implications for health promotion programmes are discussed, particularly the need to place more emphasis on tackling fathers' smoking. Currently, fathers' smoking receives far less research or health promotion attention than mothers' smoking. Protecting infants from fathers' as well as mothers' smoking is key to reducing environmental tobacco exposure in early infancy, when the risk of Sudden Infant Death is highest.

SIDS-07034

Indiana infant mortality report: 2002 period linked birth/infant death data set.

Rahmanifar A
Indianapolis, IN : Indiana Epidemiology Resource Center , 2004, 16 p.

This report presents 2002 period infant mortality statistics in Indiana from the linked birth/Infant death data set (linked file) by race, a variety of infant and maternal characteristics and leading causes of death.

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SIDS-07035

Indiana infant mortality report: 1999 period linked birth/infant death data set.

Rahmanifar A
Indianapolis, IN : Indiana Epidemiology Resource Center , 2001, 16 p.

This report presents 1999 period infant mortality statistics in Indiana from the linked birth/infant death data set (linked file) by race, a variety of infant and maternal characteristics and leading causes of death.

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