SES15.4

International guidelines for smoking cessation – an overview

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The first evidence based smoking cessation guidelines were published in 1996 by the Agency for Health Care Policy and Research (Smoking Cessation: Clinical Practice Guideline. JAMA. 1996; 275:1270–1280). Since then a number of national guidelines and international have been developed (e.g. World Health Organization. First WHO Recommendations on the Treatment of Tobacco Dependence. WHO Regional Office, Copenhagen, 2001; Fiore MC, Bailey WC, Cohen SJ, et al. Treating Tobacco Use and Dependence. Clinical Practice Guideline. Rockville, MD: U.S. Department of Health and Human Services. Public Health Service, 2000; Arzneimittelkommission der Deutschen Ärzteschaft: Therapieempfehlungen Tabakabhängigkeit. Arzneiver-ordnungen in der Praxis, Sonderheft, 2001). Evidence based smoking cessation guidelines have summarised the main treatment recommendations proposing that maximum support in motivating the smoker should be offered. If otherwise impossible, this should take the form of brief contacts. More complex interventions should be supplemented by behaviour therapy aimed at changing of the behaviour patterns. Medical support should be offered using nicotine replacement (NRT) or bupropion. Moreover international guidelines, American, and European, as well as German guidelines recommend combining smoking cessation methods based on behavioural treatment with nicotine replacement therapy or bupropion as the most effective strategy.

SP02. Round table discussion on education in Europe

Chairs: M. Musalek (A), A. Lindhardt (DK)
Participants:
Paul Cosyns (B)
Anne Lindhardt (DK)
Michael Musalek (A)
Henning Sass (D)
Sam Tyano (IL)

S60. Alcohol and the developing organism – fetus, child and youth

Chairs: U. Rydberg (S), E. Halmesmäki (FIN)

S60.1

The fetus and external agents

I. Krägeloh-Mann. *Germany*

No abstract was available at the time of printing.

S60.2

Alcohol and neurotrophic factors during pregnancy

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Approximately 1–5% of pregnant women in Finland abuse alcohol. The corresponding figure for drug abuse is some 0.3–0.5%. Both alcohol and drugs pass the placenta easily, and both alcohol and drugs can be measured from amniotic fluid, fetal tissues, newborn urine and meconium. The amount and way of use of drugs (orally, intravenously, inhalation) have an impact on both the maternal and fetal concentrations, those achieved intravenously being the highest.

Fetal alcohol syndrome, FAS, is well known since 1978. The diagnostic criteria include pre- and postnatal growth retardation (microcephalia, low birth weight and length), neurological symptoms and/or mental retardation, abnormal facial characteristics. During pregnancy, the growth of uterus is impaired already by the 30th gestational week, and by ultrasound scanning one of the main FAS features, microcephalia, is possible to diagnose in subjects with severe abuse by that time as well. During maternal alcohol intoxication the fetal cardiotocography is often pathological, but normalizes when the effect of alcohol is going over. At birth, these infants are irritated, do not eat (they may need a feeding tube for several weeks), and may have neurological symptoms and an abnormal EEG.

The infants of drug addicts may suffer from severe withdrawal syndroms, including low blood sugar, irritation, poor feeeding, apnea, vomiting etc. Sometimes the infant needs to have morphine for these symptoms. However, not such a clear neonatal syndrome as FAS can be definied in infants of drug addicts, maybe because the fetal exposure, amount and timing, and type of drugs used vary individually so much.

S60.3

Excitatory and inhibitory neuroreceptors: their role for the fetal alcohol syndrome

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Reduction of brain mass and of the functional capacity of the brain are two main features associated with the neurotoxic syndrome caused by intrauterine exposure of the human fetus to alcohol, an event, which results in fetal alcohol effects (FAE) and/or the fetal alcohol syndrome (FAS). Preclinical findings have not been able to fully explain how the demonstrated limited neuronal loss in certain brain areas could account for the typical overall reduction in brain mass or for the complex neurobehavioral disturbances observed in FAE/FAS children. More recent evidence suggests that a blockade of NMDA/glutamate receptors and/or an overactivation of GABA receptors during synaptogenesis (last trimester of gestation) may trigger apoptotic neurodegeneration in the developing brain and that there is a well-defined time window for the demonstrated vulnerability to this excessive stimulation of NMDA/GABA receptors in the immature brain. Since alcohol inhibits NMDA produced excitation and enhances GABAergic neurotransmission it is suggested that these effects of alcohol in the immature brain may well explain the variety of neurobehavioral disturbances and the morphological changes occurring later in life.

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