SMOKING AND HEALTH

- D.Hrubá
- https://is.muni.cz/www/2422/um

- SMOKING IS THE MOST IMPORTANT SINGEL PREVENTION OF MORBIDITY AND MORTALITY - WHO

More people will die

- FROM SMOKING Every year ,
- Than from : murder, AIDS, suicide, drugs, car crashers and alcohol

  COMBINATED

SMOKING CONTRIBUTES TO

- MORTALITY: annually 6 mil. victins worlwide
- MORBIDITY: at least to 25 different diseases
- DISABILITY: the second leading factor

GLOBAL BURDEN of DISEASE

- ATTRIBUTABLE EFFECTS of 67 RISK FACTORS in 21 WORLD REGIONS
- On DALYs (disability-adjusted life years)
- On YLD (sum of years lived with disability)
- On YLL (years of life lost)

FIVE LEADING FACTORS:

- (1) HYPERTENSION
- (2) TOBACCO SMOKING
- (3) ALCOHOL USE
- (4) DIETARY – MALNUTRITION
- (5) PHYSICAL INACTIVITY

TYPES OF TOBACCO PRODUCTS

- COMBUSIBLE: CIGARETTES, PIPES, CIGARS
- BIDIS, KRETEKS, STICS, WATER PIPES,
- SMOKELESS: SNUFS (MOIST, DRY), CHEWING TOBACCO
- ELECTRONIC NICOTINE DELIVERY SYSTEMS (vaporised liquid: e-cigarettes, pipes…, JUUL)
- HEATED-NOT-BURN TOBACCO PRODUCTS (IQOS – PM),

CHANGES

- WHILE 20th CENTURY WAS THE CIGARETTE CENTURY
- 21st CENTURY SEEMS TO BE e-CIGARETTE/ ENDS (electronic nicotine delivery systems) CENTURY

SMOKING AND HEALTH

SMOKING IS THE MAIN SINGLE PREVENTABLE FACTOR,

ALL FORMS OF TOBACCO ARE ADDICTIVE AND LETHAL

https://is.muni.cz/www/2422/um

IN CIGARETTE SMOKE

- ABOUT 5.000 CHEMICALS with
- IRRITATION,
- TOXIC,
- CARCINOGENIC,
- TERRATOGENIC, EMBRYOTOXIC EFFECTS

USING TOBACCO HAS:

- IMMEDIATE EFFECTS
- MIDLE- TERM EFFECTS
- LONG-TERM EFFECTS

- ON HUMAN HEALTH

* *IMMEDIATE EFFECTS*

- ACTIVATION OF BRAIN RECEPTORS
- CARDIOVASCULAR CHANGES
- HYPOXEMIA
- IRRITATION

NICOTINE IN BRAIN

- NICOTINE REACHES THE BRAIN WITHIN 10-20 SECONDS AFTER THE PUFF,
- WITHIN 20-30 MINUTES AFTER TRANSDERMAL/SALIVA TRANSPORT
- NICOTINE OCCUPIES THE SPECIFIC CHOLINERGIC RECEPTORS AND INDUCES THEIR ACTIVATION

ACETYLCHOLINE RECEPTORS - nAChRs

- TWO UNITES: ALPHA, BETA
- SEVERAL SUBUNITES
- PRESENT ON NEURAL CELLS (both central and peripheral), and
- ON TISSUE CELLS
- SOME SUBUNITES ARE NICOTINE SPECIFIC (activated by nicotine)

DENSITY of nAChRs

- IN THE BRAIN IS NOT HOMOGENOUS
- ACCUMULATION IN N. ACCUMBENS =>
- LIMBIC AREA
- AFTER THEIR ACTIVATION => SERIES OF PHYSIOLOGICAL EVENTS

RELEASE OF NEUROTRANSMITTERS:

- DOPAMINE
- SEROTONINE
- ACETYLCHOLINE
- EPINEPFRINE, NOREPINEPHRINE,
- BETA-ENDORPHINE
- ACTH, ADRENALINE

EFFECTS OF NICOTINE

- WELL BEEING (DOPAMINE)
- COPING THE STRESS (ACTH)
- BETTER SHORT-TERM PERFORMANCE (ACETYLCHOLINE, ADRENALINE)

THESE EFFECTS

- CAN PRODUCE MANY NATURAL DAILY EVENTS:
- FOOD, SEX
- MUSIC, SUCCESS
- FRIENDLY ENVIRONMENT

DUE TO THESE REWARDS

- SMOKERS REPEATE PUFFS,
- RAISE THE NUMBER DAILY SMOKING CIGARETTES
- LIGHT ON AUTOMATICALY IN SPECIFIC SITUATIONS
- DEVELOP ADDICTION

TWO FACES OF TOBACCO COMPANIES

- „NICOTINE IS THE ADDICTING AGENT IN CIGARETTES“

Private statement, Brown &Williamson official in 1983

„I BELIEVE THAT NICOTINE IS NOT ADDICTIVE“

Sworn testimory before the US Congress;

CEOs of the seven leading tobacco companies in 1994

WHAT IS TRUE?

- ALL FORMS OF TOBACCO CAN DEVELOP AN ADDICTION
- THE DRUG IS NICOTINE
- ITS PATHWAYS AND POWERTY IS SIMILAR AS THOSE OF HEROINE and COCCAINE

US.Surgeon General Report, 1988

OFFICIAL STATUS

- Dg. F 17:

PSYCHOLOGICAL AND BEHAVIORAL DISORDERS CAUSED BY TOBACCO USE

International statistic classification of diseases, 10th revision, 1991

SMOKING ADDICTION

- 80 – 85% OF CURRENT SMOKERS WILL BE DEPENDENT, SIMILARLY LIKE CURRENT USERS OF HEROINE OR COCCAINE
- ABOUT ONE THIRD OF OCCASSIONAL SMOKERS WILL BE DEPENDENT

SMOKING IS A DISEASE

- DEPENDENCE ON SMOKING IS NOT A LACK OF WILLING OR „BAD HABIT“ BUT
- CHRONICAL, PROGRESSIVE AND RELAPSING DISEASE
- BOTH PHARMACOLOGICAL AND BEHAVIORAL ADDICTION

ALTERED DOPAMINERGIC SYSTEM

- PREMATURAL ACTIVATION OF FETAL RECEPTORS
- DECREASED AMOUNT OF NEURAL CELLS IN THE BRAIN
- SUDDEN INFANT DEATH SYNDROME
- IMPAIRED NEURO-PSYCHOLOGICAL DEVELOPMENT
- BEHAVIORAL and COGNITIVE PROBLEMS

ALTERED SEROTONERGIC SYSTEM

- MAJOR PSYCHIATRIC DISORDERS (SCHIZOPHRENIA, DEPRESSION)
- 2-3 times HIGHER FREQUENCY OF SUICIDES
- SMOKING CAUSES DEPRESSION
- DEPRESSION CAUSES SMOKING

CARDIOVASCULAR CHANGES

- VASOCONSTRICTION: SKIN, CORONARY, BRAIN, ABDOMINAL, VERTEBRAL, PLACENTAL ARTERIES
- HIGHER BLOOD PRESSURE
- HIGHER HEART RATE
- HIGHER HEART VOLUME/MIN
- DECREASED SKIN TEMPERATURE

MECHANISMS OF ACTION

- ACTIVATION OF SYMPATIC NERVOUS SYSTÉM
- RELEASE OF SUPRARENAL HORMONES (ADRENALINE, NORADRENALINE)
- BY QUICK ADMINISTRATION OF NICOTINE

VASOCONSTRICTION

- CONTINUE EVEN AFTER THE CIGARETTE IS SMOKED
- FOR ANOTHER 30 – 45 min
- AS NICOTINE IS PRESENT IN BLOOD

HYPOXEMIA

- DECREASED AMOUNT OF BLOOD DUE TO VASOCONSTRICTION (caused by nicotine)
- DECREASED AMOUNT OF OXYGEN IN BLOOD (caused by carbon monoxide – COHb)
- DECREASED BLOOD-TISSUE TRANSPORT OF OXYGEN (caused by hydrogen cyanid HCN)

IN PREGNANCY

- LOCAL PLACENTAL NECROSIS (caused by cadmium Cd)
- POWERFULL AFFINITY OF FETAL HEMOGLOBIN TO CARBON MONOXIDE ENHANCES COHb LEVELS BY 25% (fetal x maternal blood)

OXIDATIVE STRESS

- IN PLACENTAL TISSUE IMPAIRES DEVELOPMENT AND FUNCTION DUE TO DAMAGES OF DNA,
- INCREASING OF APOPTOSIS AND
- CELLULAR DEATH

HYPOXEMIA and HYPONUTRITION

- FETAL GROWTH RETARDATION = FETAL TOBACCO SYNDROME =>
  programing of OBESITY, LIPID AND GLUCOSE METABOLISMS
- =>RISE RISK of CVD
- RISK OF PRE-TERM BIRTH
- RISK OF INTRAUTERINE DEATH

PRENATAL EXPOSURE

* ALTERES THE LUNG DEVELOPMENT
- MODIFIES THE REPRODUCTIVE DEVELOPMENT
- RISES THE RISK OF CONGENITAL MALFORMATIONS, and
- SUDDEN INFANT DEATH SYNDROME

HYPOXEMIA IN ADULTS

- HEART ATTACK (IM)
- CEREBROVASCULAR ATTACK (STROKE)
- WRINKLING, PREMATURE AGEING
- IMPAIRED WOUND HEALING
- LEG AND HAND PAIN, GANGRENE – PERIPHERAL VASCULAR DISEASE

IRRITATION

- EYES:excessive tearing, blinking, stinging
- NOSE: bad smell, stinging, phlegm
- NASOPHARYNX: cough, cold in the chest

- STRESS DUE TO DYSCOMFORT

* *SHORT/MILD-TERM EFFECTS*

- IMPAIRED IMMUNITY
- HORMONAL DYSBALANCE
- IMPAIRED BLOOD LIPIDS
- IMPAIRED HEMOCOAGULATION
- CHRONIC INFLAMMATION

IMMUNE SYSTEM

- IMPAIRED RESISTANCE TO INFECTION
- CONTRIBUTION TO ALLERGIES
- INFANTS AND CHILDREN ARE THE MOST VULNERABLE POPULATION
- IMPAIRED RESISTANCE TO CANCER (Natural Killers)

MALE REPRODUCTION

- IMPOTENCE (erectile dysfunction)
- IMPAIRED SPERMIOGENESIS: deformity, loss of motility, reduced number, aneuploid sperm cells
- FETAL MALFORMATIONS
- INFERTILITY

ERECTILE DYSFUNCTION (ED)

- PERSISTENT / RECURRENT INABILITY TO ACHIVE/MAINTAIN AN ERECTION SUFFICIENT FOR SATISFACTORY SEXUAL PERFORMANCE (1993)
- 20 % of all men, 52 % in age 40-70 y.

SMOKING INCREASES ED

- FROM 2005 (Austoni et al.) studies in many populations: China, Middle East, Europe, America)
- OR = 1,4 – 3.1 with respect to other causes of ED
- Smoking effects on ED are dose dependent

MOLECULAR MECHANISMS

- PARASYMPATHETIC NERVOUS SYSTÉM INDUCES SMOOTH MUSCLE RELAXATION => ALLOWS ARTERIAL PRESSURE BLOOD INTO THE CORPUS CAVERNOSUM VIA ACTION of NITRIC OXIDE (NO)
- NICOTIN IS SYMPATICOMIMETIC,
- LEVELS OF NO – are altered by smoking

SMOKING CESSATION

- NO CONSISTENT RESULTS ABOUT THE MAGNITUDE OF THE BENEFITS WITH REGARD TO ED
- HISTORY OF SMOKING PRODUCE SILENT VASCULAR INSULT THAT PERSIST OVER TIME

BETTER PROGNOSIS

- IS BELIEVED FOR YOUNGER MEN WITHOUT SO LONG SMOKING HISTORY and
- WITH LACK OF COMORBIDITIES.

- Kovac JR et al. Effects of cigarette smoking on erectile dysfunction
- Andrologia 2014;
- Doi: 10.1111/and.12393

FEMALE REPRODUCTION

- PAINFUL MENSTRUATION
- EARLIER MENOPAUSE
- INFERTILITY
- ECTOPIC PREGNANCY
- PLACENTA PRAEVIA
- PREMATURE BIRTH
- SPONTANEOUS ABORTION

BOTH ACTIVE AND PASSIVE SMOKING ARE RISKS (OR):

• ACTIVE SMOKERS:	• PASSIVE SMOKERS
• ABORTIONS ….1.16	• ……..1.17
• STILLBIRTHS…1.44	• ……. 1.55
• ECTOPIC PR. … 1.43	• ……. 1.61

SMOKING

- SEEMS TO BE PROTECTIVE for the development of
- PREECLAMPSIA
- BUT IT IS NOT A REASON FOR KEEPING SMOKING DURING PREGNANCY!!!, AS OTHER RISKS OVERTOP THIS BENEFIT

OTHERS

- HORMONAL DYSBALANCE CONTRIBUTES TO:
- DIABETES MELLITUS and COMPLICATIONS
- OSTEOPOROSIS and
- HIP FRACTURES
- By the ANTIESTROGENIC EFFECT OF NICOTINE

BLOOD LIPIDS

- INCREASED LEVELS OF
- TOTAL CHOLESTEROL
- LDL – CHOLESTEROL
- VLDL – CHOLESTEROL

- DECREASED LEVELS OF
- HDL- CHOLESTEROL

HEMOCOAGULATION

- ENHANCED ACTIVITY OF THROMBOCYTES and
- FACTOR VIII =>

- ARTERIAL THROMBOSIS (IM, stroke, gangrene in perifery)

SMOKING IS RESPONSIBLE

- FOR 25% OF ISCHEMIC HEART D.
- FOR 25% OF VASCULAR DISEASES (stroke, Burger d., aneurysma, macular degeneration, cataracts)
- FOR EARLIER ATHEROSCLEROSIS
- FOR 75% OF CHRONIC OBSTRUCTIVE PULMONAL DISEASE (chr. Bronchitis, emphysema)

EXPOSURE TO ETS

- INCREASES THE RISK OF Ac.coronary sy by 25 – 30 %:
- INCREASED THROMBOGENESIS and
- LDL OXIDATION
- DECREASED ABILITY TO EXERCISE
- ACTIVE INFLAMMATORY PATHWAY.
- IMPAIRED VASCULAR REPAIR

SMOKING CONTRIBUTES TO

- STOMACH AND DUODENAL ULCERS
- TEETH LOOSE
- GUM DISEASES – GINGIVITIS, PERIODONTITIS
- PROGRESSION OF PRESBYACUSIS
- PSORIASIS and other skin diseases
- TREMOR

MENTAL HEALTH:

- Smoking negative influences on:
- - brain development,
- - memory

Smoking is a risk factor for Alzheimer disease

Smoking is a protective factor for Parkinson disease

* *LONG-TERM EFFECTS*

- TOBACCO SMOKE CONTAINS OVER 5.000 CHEMICALS,

- 67 OF WHICH ARE CONFIRMED or SUSPECTED HUMAN CARCINOGENS (within the list of appr. 73 confirmed human chemical carcinogens)

CARCINOGENS IN SMOKE

- POLYCYCLIC AROMATIC H. (benzo/a/pyrene)
- HEAVY METALS (Cd, As)
- RADIOACTIVE POLONIUM 210
- INDUSTRIAL CARCINOGENS:beta-naphthylamine, 4-aminobiphenyle, benzene, formaldehyde

TOBACCO SPECIFIC NITROSAMINES

- NNK: 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone
- NNAL
- NNN
- And many others

SMOKING IS RESPONSIBLE

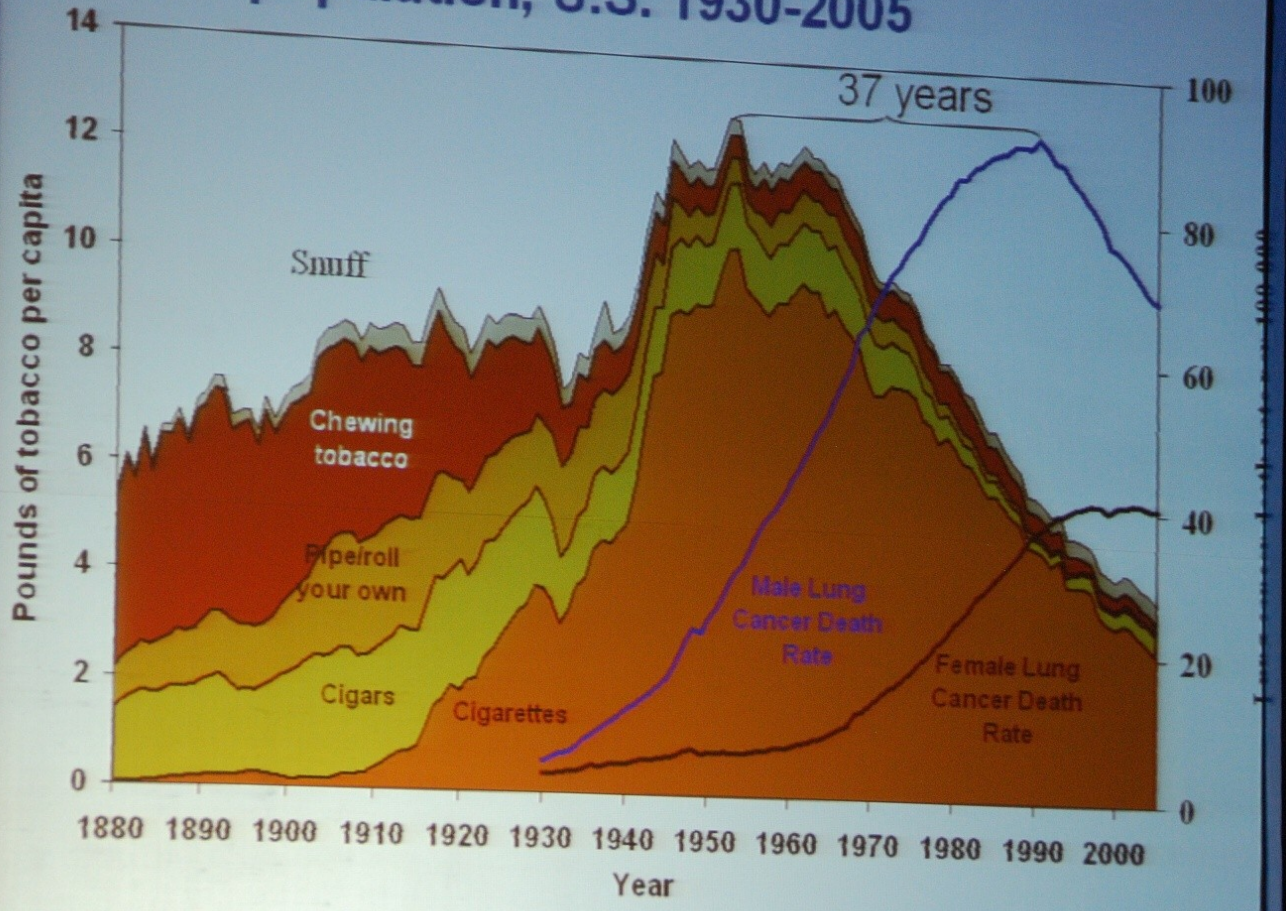
- FOR 90-95% OF ALL LUNG CA
- FOR 40-60% OF HEAD/NECK CA
- FOR 40-60% OF KIDNEY/BLADDER CA
- FOR 30% OF CERVICAL CA
- FOR 30% OF GASTRIC/PANCRETIC CA
- FOR COLON, LIVER, BREAST CA

LUNG CARCINOGENS

- ACTIVE SMOKING ……. 90 %
- PASSIVE SMOKING …… 4 %
- OCCUPATION …… 5 %

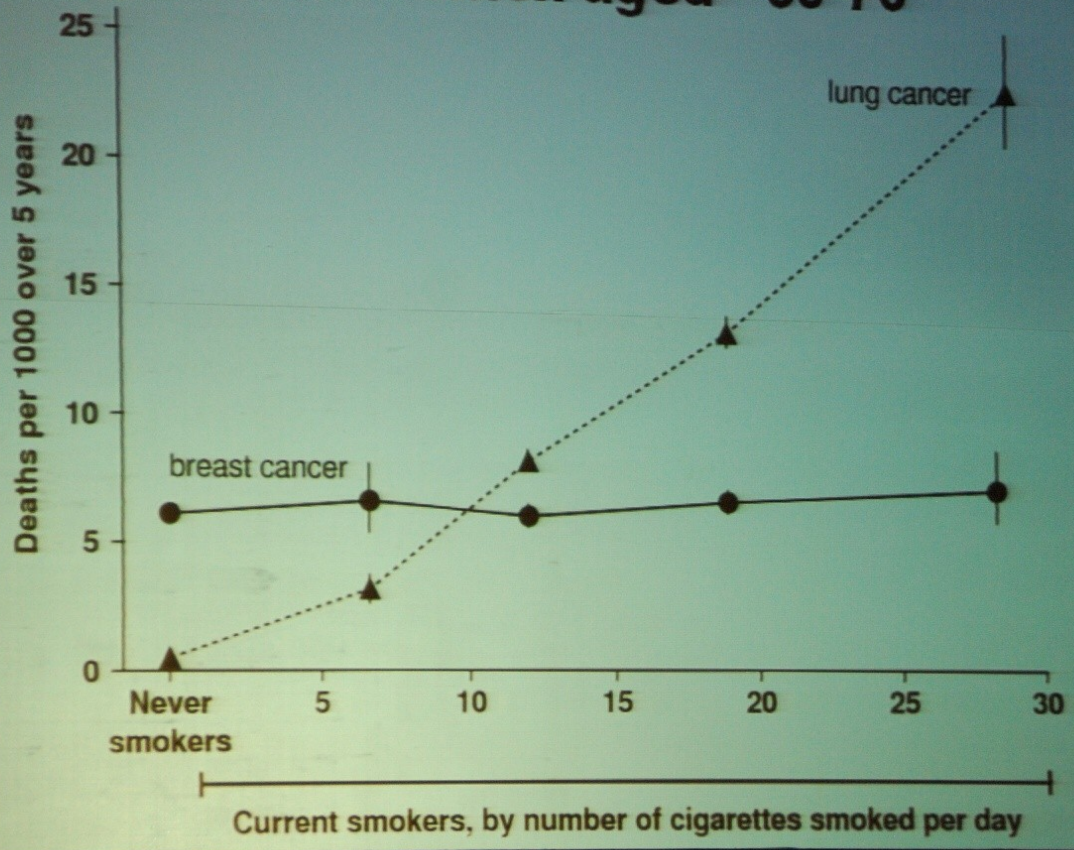
  (radiation, asbestosis, Cr, Ni, PAH, plast additives, benzin…)
- ENVIRONMENT ……. . 1 %

Lung cancer death rates in men & women: General population, U.S. 1930-2005



THE MILLION WOMEN STUDY

LUNG CANCER & BREAST CANCER MORTALITY
per 1000 women aged ~50-70



PATHWAYS:

- GENOTOXICITY => INITIATION OF CARCINOGENESIS
- METABOLIC ACTIVATION – microsomal enzymes P 450 – HEREDITARY DETERMINATION
- EPIGENETIC EFFECTS =>MODULATE CELLULAR FUNCTIONS => TUMOR PROMOTION and PROGRESSION

CARCINOGENESIS:

- GENOTOXIC EFFECTS:

1. INITIATION of DNA MUTAGENIC CHANGES
2. REPLICATION

- EPIGENETIC EFFECTS:

  INFLUENCE ON APOPTOSIS

1. PROMOTION
2. PROGRESSION
3. METASTASES

GENOTOXIC CARCINOGENS

- NITROSAMINES - NNK
- PAU – BENZO/A/PYRENE
- RADIOACTIVITY - Po 210
- INDUSTRIAL CARCINOGENS - ACROLEIN, 4-AMINOBIFENYLE, BETA-NAFTYLAMINE, BENZENE

CONSEQUENCES: PROMOTION

- CELL´S PROLIFERATION
- ANTI - APOPTOSIS
- PROTEIN SYNTHESIS
- MITOCHONDRIA DYSFUNCTION
- INCREASING of REPLICATIVE LIFESPAN of CARCINOGENIC CELLS

CONSEQUENCES: PROGRESSION and INVASION

- ANGIOGENESIS =>
- SUPPORT FOR THE TUMOR GROWTH
- DECREASED CELLS´ ADHERENCE =>
- METASTASIS

EPIGENETIC CARCINOGENIC ACTIVITIES

- MEDIATED THROUGH nAChR WERE FOUND FOR:
- NICOTINE
- NNK
- POLYCYCLIC AROMATIC HYDROCARBONS

nACh RECEPTORS:

- CELL – TYPE – SPECIFIC
- MODIFIED BY VARIOUS ENVIRONMENTAL FACTORS
- UNDERSTANDING of MOLECULAR MECHANISMS => FUTURE DEVELOPMENT IN CANCER DIAGNOSES/THERAPIES

ROLE OF microRNAs

- PROBABLY MORE THAN 1000
- KNOWN MORE THAN 700
- TISSUE SPECIFIC
- DETECABLE IN 12 BODY FLUIDS
- SOME OF THEM EARLIER MARKERS OF CARCINOGENIC CHANGES

SMOKING KILLS

- HALF OF ALL LIFETIME USERS
- HALF OF THEM WILL DIE BETWEEN
  30-69 YEARS OF AGE
- IN THE 20th CENTURY,
  100 MILLION PEOPLE
  DIED FROM TOBACCO USE

SMOKING KILLS

IN 2000

- 4,8 MIL ANNUAL PREMATURE DEATH
- 3,8 MILLION MEN
- 1,0 MILLION WOMEN

BY 2020 TOBACCO WILL KILL ABOUT

- 10 MILLION PEOPLE EVERY YEAR

SMOKING KILLS

- TOBACCO WILL KILL

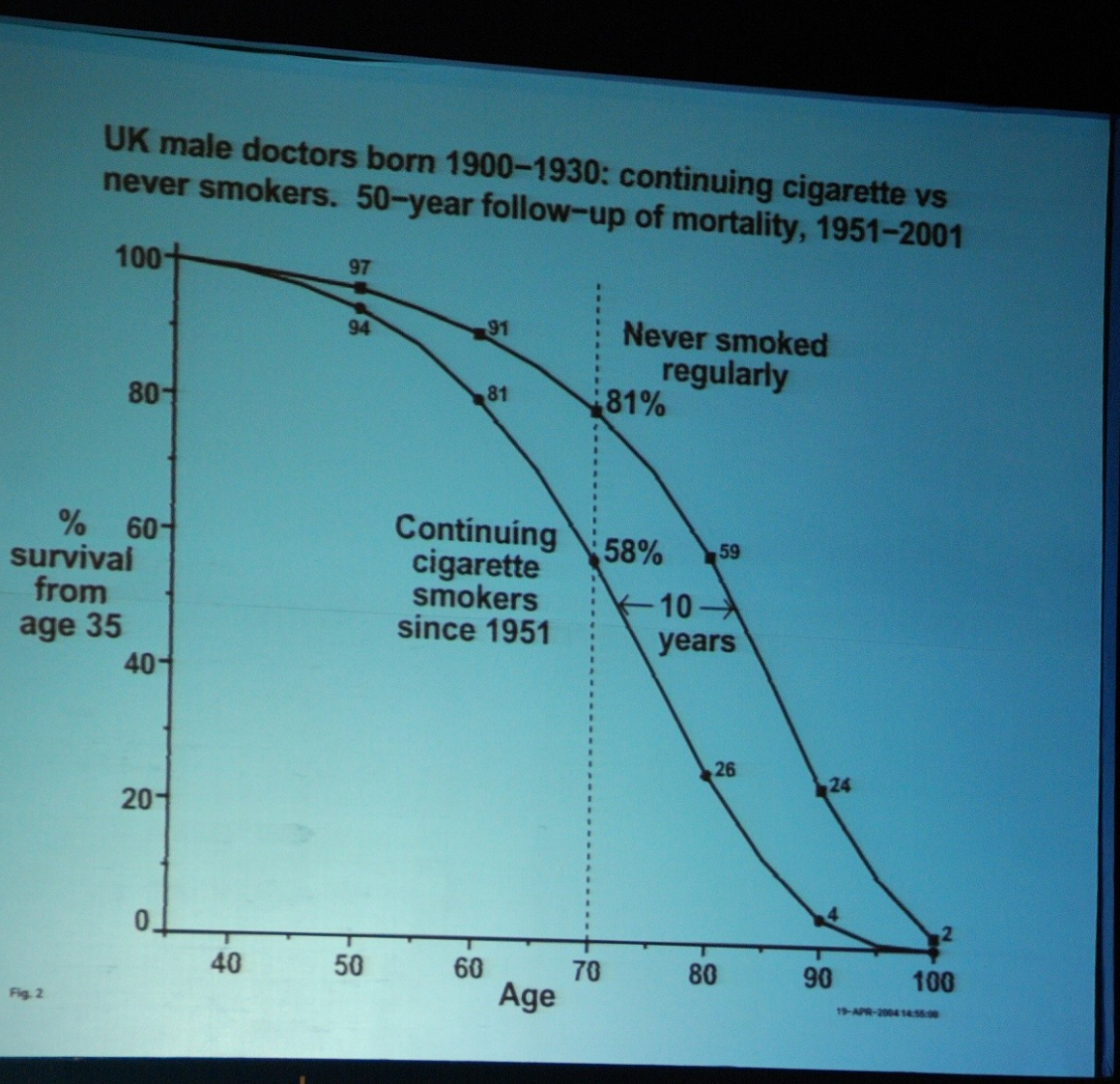
  1 BILLION = 100 000 000

  PEOPLE

- IN THE 21st CENTURY

SMOKING KILLS PHYSICIANS

- British Medical Doctors Study (Doll, Lopez, Peto): smokers lost
- 5 YEARS OF LIFE - 1951-1971
- 8 YEARS OF LIFE – 1971-1991
- 10 YEARS OF LIFE – 1991-2006


UK male doctors born 1900–1930: continuing cigarette vs never smokers. 50–year follow–up of mortality, 1951–2001
100
80
60
40
20
0
% survival from age 35
97
94
91
81
Never smoked regularly
81%
Continuing cigarette smokers since 1951
58%
59
10 years
26
24
4
2
40
50
60
70
80
90
100
Age
Fig. 2
19-APR-2004 14:55:00

SMOKING KILLS NON-SMOKERS

- MAIN STREAM
- 800-900^{o} C
- 16% O_2
- 6,0-6,7 pH

- SIDE STREAM
- 600^{o} C
- 2% O_2
- 6,7-7,5 pH

DANGER FOR NO-SMOKERS

- SECONDHAND SMOKE
- ENVIRONMENTAL TOBACCO SMOKE
- PASSIVE SMOKING
- INVOLUNTARY SMOKING

Side stream + smoker´s expiration + chemicals interaction

SIMILARITIES and DIFFERENCES

- THE NUMBER OF CHEMICALS in MS and SS ARE THE SAME
- THE LEVELS OF CHEMICALS ARE HIGHER in SS COMPARRED TO MS
- DUE TO IMPERFEKT BURNING

RATIO SS : MS - IRRITANTS

- ACROLEIN 8 – 15
- FORMALDEHYDE 10 – 15
- AMONIUM 73
- NITROGEN OXIDES 4 – 10
- FORMAMIC ACID 1,5
- NAFTALENE 16

RATIO SS : MS - TOXINS

- CARBON MONOXIDE 2 – 5
- TOLUENE 6 – 8
- NICOTINE 2,6-3,3
- NICKEL 13 – 30
- POLONIUM 210 1 – 4
- PCDD, PCDF 2

RATIO SS : MS - CARCINOGENS

- BENZENE 5 – 10
- NITROSAMINES 20 – 100
- 2-NAFTYLAMINE 30
- 4-AMINOBIFENYLE 30
- BENZO/A/PYRENE 2,5 – 3,5
- TAR 1,7

INDOOR CONCENTRATIONS OF NICOTINE

- WORK-PLACES 20 ug/m^3
- CONFERENCE HALL 40 ug/m^3
- RESTAURANTS 26-28 ug/m^3
- CARS 40 ug/m^3
- HOMES 7-11 ug/m^3
- HOSPITALS 0,01- 4 ug/m^3

INDOOR CONCENTRATIONS OF NITROSAMINE NNK

- BARS 10 – 24 ug/m^3
- RESTAURANTS 1 – 3 ug/m^3
- TRAINS 5 ug/m^3
- CARS 29 ug/m^3
- OFFICES 26 ug/m^3
- HOMES 2 ug/m^3

THIRDHAND SMOKE

- SOME CHEMICALS IN ETS ARE ABSORBED IN WALLS, CARPETS, CLOTHES, FORNITURE
- AND ARE RE-EMITTED INTO THE INTERIER

THIRDHAND SMOKE

- NICOTINE + NITRIC ACID + NO_X
- = > INTERACTIONS = >
- NITROSAMINES NNK, NNA, NNN
- (mutagenic, carcinogenic)
- CONTAMINATION OF CLOTHES, SKIN, CARPETS, FORNITURE for many hours, days, weeks, years

1st EXPERIMENTAL STUDY:

- MICES EXPOSED TO THIRDHAND S.
- LIVER: FIBROSIS, STEATOSIS
- BLOOD: INCREASED TOTAL and LDL CHOLESTEROL, DECREASED HDL CHOLESTEROL
- PRE-DIABETIC DEFECTS OF INSULIN METABOLISM
- LUNG: FIBROSIS, INCREASED NUMBER of MACROPHAGES => OXIDATIVE STRESS
- SKIN: KERATINOSIS, LESS FIBRILLAR COLLAGEN
- HYPERACTIVITY, ANXIETY

EXPOSURE TO ETS - CHILDREN

- UNPLEASANT DYSCOMFORT
- IRRITATION
- IMPAIRED IMMUNITY
- RESPIRATORY INFECTIONS, ALERGY
- SIDS
- LEUKEMIA, BRAIN TUMORS

EXPOSURE TO ETS - ADULTS

- UNPLEASANT DYSCOMFORT
- IRRITATION
- ACCUTE CORONARY ISCHEMIA
- CHRONIC OBSTRUCTIVE PULMONARY DISEASE
- LUNG CANCER

HEALTH CONSEQUENCES OF EXPOSURE TO ETS:

- AN HOUR A DAY IN A ROOM WITH SMOKER
- IS NEARLY A HUNDRED TIME MORE LIKELY TO CAUSE LUNG CANCER IN A NON-SMOKER
- THAN TWENTY YEARS SPENT IN A BUILDING CONTAINING ASBESTOS

Sir Richard Doll, 1989

BAN OF SMOKING ON PUBLIC PLACES

- 18 months after implementation:
- INCIDENCE of MI DECLINED BY 33%
  from 150.8 to 100.7 / 100.000
- INCIDENCE of SUDDEN CARDIAC DEATH DECLINED BY 17%
  from 109.1 to 92.0 / 100.000

Minnesota Study , Arch.Intern.Med. Doi: 10.1001/2013.jamainternmed.46

SMOKE-FREE LEGISLATION

- LOWERED RATES OF HOSPITAL ADMISSIONS for: RR
- CORONARY EVENTS …… 0.848
- OTHER HEART DIS. …… 0.610
- CEREBROVASCULAR DIS. … 0.840
- RESPIRATORY DIS. …… 0.760

Circulation 2012; 126: 2177 - 2183

ANTENATAL EXPOSURE

- GROWTH RETARDATION
- DELAYED LUNG DEVELOPMENT
- ACTIVATION OF nAChs (by NICOTINE) = NEUROTERRATOGENICITY (CONDUCT DISORDERS, ADHD, REDUCED MENTAL / SCHOOL PERFORMANCES)

PRENATAL PROGRAMING

- EXPOSURE TO MATERNAL SMOKING
- => CHANGES IN FETAL METABOLISM
- OUTLAST AFTER DELIVERY
- => OBESITY, HYPERTENSION, DAMAGES SERUM LIPIDS´ RATES in CHILDHOOD and ADULTHOOD

CONCLUSSION

- CIGARETTE IS UNIQUE ARM KILLING BY ITS BOTH ENDS
- GLOBAL TOBACCO EPIDEMY IS WORSE TODAY THAN 50 YEARS AGO AND MAY BE WORSE IN ANOTHER 50 YEARS
- SMOKING IS THE MOST IMPORTANT PREVENTABLE RISK FACTOR