TOBACCO SMOKING

Anupam Prakash
Department of Medicine
Lady Hardinge Medical College,
New Delhi
TOBACCO SMOKING

- Historical perspective
- Burden of smoking
- Cigarette manufacture
- Cigarette smoke
- Deposition & absorption of smoke
- Quantification of tobacco exposure
- Effects of smoking – active & passive
- Smoking cessation
- Success stories & Alternative avenues
• **Nicotine molecule** – produced over 60 million yrs; probably to guard against insect behaviours.

• Antiquity of smoking - 692 AD – Mayan priest smoking

• 16th century – tobacco cultivation spread by Spanish & Portuguese sailors

• 17th century – pipe smoking and snuff were dominant - declined in 19th century
HISTORICAL PERSPECTIVE

• **Precursors of cigarettes**
  - Aztecs smoked long tobacco filled reeds
  - Spaniards refined technique with use of paper tubes
  - 1870s handmade cig replaced by machine made ones
  - Between the world wars cig. smoking gained popularity
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TOBACCO USE IS A RISK FACTOR FOR SIX OF THE EIGHT LEADING CAUSES OF DEATH IN THE WORLD

WHO report on the global tobacco epidemic, 2008
• 1.3 billion smokers
• Approx 50 LAKHS people die from tobacco use every year (1 death every 6 seconds)- equal in dev & devg countries
• Tobacco smoking including second hand smoke- 2nd leading risk factor for global disease burden
• Tobacco kills up to half of its users

WHO report, 2011
Burden of smoking - India

- Home to 1/5 of 1.3 billion world tobacco users
- 1/3 of all tobacco users in the developing world
- 600,000 die every year (15% of global tobacco deaths)
- 55000 Indian children addicted every year
- 40% men light regularly
- 50% will die from tobacco-related diseases
- Little change in habits in India
THERE IS AN EASIER WAY OF COMMITTING SUICIDE

TRY SMOKING
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Cigarette manufacture

Tobacco plant
sprayed with ‘casing’
sauce of sugars, humectants, flowering agents
harvested

Cured by air drying (burley)
Cured by artificial heat (flue curing)

Tobacco leaf then processed into sheets
Mixed with additives to improve taste
Chopped to make cigarettes

Tobacco wrapped in a tube of paper, filter of cellulose acetate at end

Filtered cigarettes less tar & nicotine, but may have same or higher yield of CO
• Each cigarette contains 1gm tobacco
• Burley tobacco yields tar 20-40 mg per cigarette
• 74 or 84mm length - regular or king size cig.
• Bidi
  – 0.5 gm dried & cured tobacco flakes, hand-rolled in a rectangular piece of tendu leaf (*Diospyros melanoxylon*)
  – Outer covering- contributes to high draw resis, ↑ CO prod.
  – High total particulate matter (dry), including carcinogenic HCs & other toxic agents CO, NH₃, HCN & phenols (cf. NF cig)
  – 85% of world’s bidi production in India
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Cigarette smoke

- **Gas & particulate phase components**
  - N, O, CO₂, CO (4%), nitrogen oxides, NH₃, nitrosamines, HCN, nitriles, volatile HCs, CH₃CHO, HCHO, acrolein
  - Particulate – aerosol of tar/nicotine
  - Tar – complex mixture of polynuclear aromatic HCs including carcinogens as non-volatile nitrosamines, aromatic amines & benzopyrene
  - **Low tar** - < 18 mg; **High tar** > 26 mg

- **Radioactive constituents** – Polonium & Lead; carcinogenic
Cigarette smoke

- Cigarette delivers on an avg – **15 mg tar & 1 mg nicotine**
- **1983; WHO Tech Rep Ser** –
  - No difference b/w filtered & non-filtered cig.
  - Indian cig. Deliver higher tar (19-27mg) & nicotine (1-1.4mg) content
  - Bidi – High tar (> 23 mg) & nicotine (1.7-3mg) levels
- **Mainstream smoke**- produced at high temperature
  - Drawn thru the butt, during puffing
  - Predominant source of exposure for the smoker
  - Has 2-5 billion particles /ml; dia. 0.2-1.0 mm; in respirable range
Sidestream smoke—produced at lower temp

- During smouldering of cigarette b/w puffs
- Principal source of ETS (Env. tobacco smoke)
- Lower combustion temperature—more distillation products, less combustion products
- Increased amount of nitrogen containing bases as distillation products
- Combustion zone of sidestream smoke less oxygen deficient—$\text{CO}_2/\text{CO}$ ratio higher
Deposition & absorption of smoke

- Tar, nicotine & CO delivery affected by the way cig smoked
  - Residual butt length
  - Butt filter nicotine
  - Smoking behaviour –
    - No. of puffs
    - Puff pressure profile
    - Puff volume
    - Depth & duration of inhalation

Tobacco rod – fractionation column; concentrating compounds towards the butt

Particulate matter produced at much higher conc towards finishing a cigarette – advised to leave longer stubs
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Quantification of tobacco exposure

- **20 gm tobacco/day for 1yr = ONE PACK YEAR**

- **Measurement of nicotine & cotinine** (RIA or GPC)
  - Urinary cotinine- indicates smoking within last 36 hr
  - Nonsmokers – no urinary nicotine, cotinine < 10 mcg%

- **Exhaled CO measurements**
  - >8 ppm strongly suggestive of smoking

- **Venous bld carboxyHb**
  - Indicates extent of recent inhalation style smoking t1/2 of < 4 hrs)
  - Mean level correlates with no. of cigarettes smoked
  - Nonsmokers < 1.7%
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Active smoking and ill-health

- Smoking is the largest preventable cause of death
- Over all mortality ratio for current male & female smokers – 1.7 and 1.3 compared with non-smokers
  - 70% and 30% greater overall death rates, respectively
- Mainstream smoke is the predominant source of exposure to the smoker
- Quitting smoking – effective for primary and secondary prophylaxis of CAD
  - 50% risk reduction with in first year of cessation
Physiological effects of smoking

Nicotine exerts its action at cholinergic receptors

<table>
<thead>
<tr>
<th>Physiological</th>
<th>Typical acute effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEG</td>
<td>Shift towards ↑↑ frequencies in relaxed individual</td>
</tr>
<tr>
<td>Sensory receptors</td>
<td>Stimulated</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>↑ PR, periph vasoconstr, small ↑ BP</td>
</tr>
<tr>
<td>Circulatory Hormones</td>
<td>Release of catechol, vasopressin, cortisol &amp; GH</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Minimal effects</td>
</tr>
<tr>
<td>Skeletal muscle</td>
<td>↓ reflexes &amp; muscle tone, ↑ finger tremor</td>
</tr>
<tr>
<td>Gut</td>
<td>↑ tone and motor activity of bowel</td>
</tr>
<tr>
<td>Body weight</td>
<td>Slight ↓ in some by ↑ BMR, less efficient food abs &amp; decreased appetite</td>
</tr>
</tbody>
</table>
Adverse pharmacological effects of smoking

### Cardiovascular effects
- ↑ Heart rate
- ↑ Blood Pressure
- ↓ Coronary blood flow
- ↓ Blood oxygen carrying capacity
- ↓ Vascular prostacyclin
- ↓ Contractility
- ↑ Periph vasc resis

### Haematological effects
- ↑ Leucocytosis
- ↑ Leucocyte activation
- ↑ Platelet activation
- ↑ Fibrinogen
- ↑ Haematocrit
- ↑ CarboxyHb
- ↑ Blood viscosity

### Metabolic effects
- ↑ Total cholesterol
- ↑ Triglycerides
- ↓ HDL-C

### Endocrine effects
- ↑ Catecholamines
- ↑ ADH
- ↑ ACTH
Smoking Damages nearly Every Organ in the Human Body
Chemistry of tobacco smoke

More than 4,000 compounds

Gaseous phase (500)
- Carbon dioxide
- Carbon monoxide
- Ammonia
- Hydrogen cyanide
- Benzene.

Particulate phase (3500)

Alkaloid
- Nicotine
- Anabasine
- Nornicotine
- Anatabine

Tar
- Includes many carcinogens
- Polynuclear aromatic hydrocarbons
- N-nitrosamines
- Aromatic amines.

Clin Dermatol 1998;16,557–564
Following chemicals are inhaled while smoking

- Acetone (used as paint stripper)
- Ammonia (found in floor cleaner)
- Toluene (found in industrial solvent)
- Butane (a type of light fuel)
- Naphthalene (found in mothballs)
- Methanol (used as rocket fuel)
- Cancer-causing substances such as naphthylamine, pyrene, vinyl chloride, urethane and toluidine
- Hydrogen cyanide (a very poisonous substance).
Immediately stimulates the release of many chemical messengers.

When a cigarette is smoked

Within 7 seconds

Nicotine-rich blood passes from the lungs to the brain

Immediately stimulates the release of many chemical messengers.

How does nicotine cause addiction?
This release is mediated through the activation of the nicotinic acetylcholine receptors (nAChRs) in the brain.

α4β2 receptor subtype is predominant in the human brain.
Smoking
Nicotine
reaches brain
Dopamine
release
Feel good
factor
Nicotine level
decrease
Pleasant feeling
disappears
Craving
for more
Smoking

Why nicotine is addictive?
Conditioning

- Conditioning means “forming a habit or getting regular”.

- The user begins to associate specific moods, situations, or environmental factors with the pleasing effects of the drug.
Conditioning is a major factor that causes relapse to drug use after a period of cessation.

Again and again in periods of stress he will want to smoke. He develops a feeling that smoking can reduce the stress. Feels pleasant and relaxed. Person smokes. During stress.
# Cardiovascular Disease

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Relative Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total CAD</td>
<td>1.7</td>
</tr>
<tr>
<td>- SCD</td>
<td>3</td>
</tr>
<tr>
<td>- AP</td>
<td>1.1-1.5</td>
</tr>
<tr>
<td>Non-fatal MI</td>
<td>3</td>
</tr>
<tr>
<td>Abdominal Aneurysms</td>
<td>4-8</td>
</tr>
<tr>
<td>Prog of HTN to malig phase</td>
<td>5</td>
</tr>
<tr>
<td>Cerebrovasc dis</td>
<td>1-1.5</td>
</tr>
<tr>
<td>RAS</td>
<td>5</td>
</tr>
<tr>
<td>Ruptured Berry Aneurysm</td>
<td>4</td>
</tr>
<tr>
<td>PVD</td>
<td>6-8</td>
</tr>
</tbody>
</table>
Chronic Obstructive Airway Disease

- **Mortality ratio for COPD i.e emphysema and chronic bronchitis comparing smokers & non-smokers ranges from 2.3 to 24.7**

- **COPD – much more prevalent in smokers**

**Prevalence of chronic bronchitis** (Malik et al, Ind J Chest Dis 1974)

<table>
<thead>
<tr>
<th>Non-smokers</th>
<th>Bidi-smokers</th>
<th>Cigarette smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>3%</td>
<td>34.6%</td>
<td>45.4%</td>
</tr>
</tbody>
</table>

- **Mortality from chronic bronchitis in male doctors who gave up smoking for over 5 years – 25% less than those who continued to smoke**
### Organ-specific carcinogens in cig. smoke

<table>
<thead>
<tr>
<th>Organ</th>
<th>Organ-specific compounds</th>
<th>Amt in 1 cig smoke</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esophagus</td>
<td>N’-nitrosonornicotine</td>
<td>140 ng</td>
</tr>
<tr>
<td></td>
<td>Nitrosopiperidine</td>
<td>0-9 mg</td>
</tr>
<tr>
<td></td>
<td>Nitrosopyrrolidine</td>
<td>1-10 mg</td>
</tr>
<tr>
<td>Lung</td>
<td>Polonium-210</td>
<td>0.03-1.3 pCi</td>
</tr>
<tr>
<td></td>
<td>Nickel compounds</td>
<td>0-600 ng</td>
</tr>
<tr>
<td></td>
<td>Cadmium compounds</td>
<td>9-70 ng</td>
</tr>
<tr>
<td>Pancreas</td>
<td>Nitrosamines</td>
<td>?</td>
</tr>
<tr>
<td>Kidney &amp;</td>
<td>Beta- naphthylamine</td>
<td>22 ng</td>
</tr>
<tr>
<td>Bladder</td>
<td>x-aminofluorene</td>
<td>present</td>
</tr>
<tr>
<td></td>
<td>x-aminostilbene</td>
<td>present</td>
</tr>
<tr>
<td></td>
<td>o-nitrotoluene</td>
<td>21 microgram</td>
</tr>
<tr>
<td></td>
<td>DibutylNitrosamine</td>
<td>0.3 ng</td>
</tr>
</tbody>
</table>
### Organ-specific carcinogens in cig. smoke

Relative risk of lung cancer *(Notani & Singhvi, 1974, Bombay)*

<table>
<thead>
<tr>
<th>All types of smokers</th>
<th>Bidi-smokers</th>
<th>Cigarette smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.45</td>
<td>2.64</td>
<td>2.23</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cancer site</th>
<th>Mortality ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung carcinoma</td>
<td>3.8-14.0</td>
</tr>
<tr>
<td>Laryngeal carcinoma</td>
<td>6-13</td>
</tr>
<tr>
<td>Oral carcinoma</td>
<td>3.0-10.0</td>
</tr>
<tr>
<td>Esophageal carcinoma</td>
<td>1.8-8.8</td>
</tr>
<tr>
<td>Urinary bladder carcinoma</td>
<td>1.4-2.9</td>
</tr>
<tr>
<td>Pancreatic carcinoma</td>
<td>1.4-3.1</td>
</tr>
<tr>
<td>Renal carcinoma</td>
<td>1.4-2.5</td>
</tr>
</tbody>
</table>
Adverse effects of smoking on pregnancy

- Infants of smoking mothers weigh on an avg 200 g less
- Infants of smoking mothers more likely to be preterm
- Increased risk of
  - IUGR
  - Spontaneous abortion
  - Foetal death
  - Neonatal death
- Maternal smoking during pregnancy may also adversely effect
  - Infant’s long-term growth
  - Intellectual development
  - Behavioural characteristics
• Mortality rate and prevalence of PUD greater
• Smoking impairs ulcer healing
• Relationship more marked for gastric rather than duodenal ulcers
• Smoking is considered to worsen nephropathy in type 2 diabetes mellitus
Occupational contaminants & exposures

• Cigarettes may function as vectors by becoming contaminated with workplace chemicals

• Potential occupational contaminants –
  – PTFE, Formaldehyde, Boron trifluoride, Organotin, Methylparathion, Dinitro-ortho-cresol, Carbaryl, Inorganic fluorides, Inorganic mercury & lead

• Cigarettes may facilitate entry into body

• Also may cause chemical transformation

• Cotton textile workers, coal miners, firefighters – greater degree of AW obstr if smokers

• Asbestos, U, Cr, Ni, As, ether & coal gas workers – if smokers, higher risk of cancer
Passive smoking

- Def. - Exposure to tobacco combustion products from the smoking of others
- ETS – derives from mainstream & side-stream smoke
- Mainstream smoke – 1st filtered by cig, then lungs
- Side-stream smoke – 85% of smoke in room
- Typical range of respirable particulates in smoking areas- café etc. 100-700 mcg/m² – 25 times of non-smoking areas
Passive smoking

- Most frequent symptom experienced by non-smokers – eye irritation (estimate 69%)
  - Headache
  - Nasal irritation
  - Cough
  - Small but sig. ↓ in pulm fn (FVC, FEV$_1$, MEF$_{50}$, MEF$_{25}$)
  - Can ppt an asthmatic attack
  - Can cause cough & dyspnoea in COPD
  - Can worsen angina
Passive smoking

• In children, parental smoking associated with –
  – Chronic cough
  – Chronic phlegm
  – Persistent wheeze
  – Respiratory infections

• Elevated mean relative risk for lung cancer in non-smokers living with smoking spouses – 1.35 (range 0.5-3.25)
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Smoking cessation

• Self care
• Clinics & groups
• Behavioural methods
• Physician advice & counselling
• Hypnosis
• Accupuncture
• Mass media & Community programmes
• Medication
Smoking cessation

• **Self care**
  - Stop smoking books
  - Quit kits
  - Audiotapes
  - Correspondence courses
  - Filter devices- reduce tar/nicotine content

• **Clinics & Groups**
  - Formal smoking cessation clinics & programmes
  - Exercise, balanced diet, lectures, behavioural modification techniques, printed materials, group discussions & videos.
Smoking cessation

- **Behavioural methods**
  - **Aversive procedures**
    - Satiation- ↑ no. of cig.s & rate at which smoked
    - Rapid smoking – inhale from cig once every 6 sec for duration of cig or till nauseated
    - Electric shock
    - Desensitisation training
    - Breath holding
    - Over-exposure to stale smoke
  - **Positive reinforcement**
    - Self- monitoring – self consciousness leads to a decrease
    - Nicotine fading – Tapering & brand-fading
Smoking cessation

- **Physician advice & counselling**
  - Routinely asking about their smoking
  - Strongly & sincerely asking to stop
  - Assisting the patient to set a quit date
  - Providing self-help material
  - Suggesting other sources of support & arranging follow-up visits

- **Hypnosis**
  - Can be helpful; who failed cessation & need intensive individual attention

- **Accupuncture**
  - placebo;
  - endorphin release which suppresses smoking withdrawal symptoms
Smoking cessation

• **Medication**
  – **Smoking deterrents** –
    • herbs, spices, mouthwashes which produce a disagreeable taste
    • Act by irritating nasal or oral mucosa or creating a dry mouth
    • Usually contain silver acetate – direct action on tongue
  – **Nicotine replacement therapy**
    • Nicoterette – chewing gum 2 mg nicotine bound to resin
      – Causes automatic reduction in smoking by 50%
      – 12-16 pieces per day, every hour while awake
      – Taper gradually after 3-6 months
      – Min. 2 months ; 6 months for majority
    • Nicotine TDS
  – Buspirone, BDZ, Clonidine, Bupropion
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Success stories

• Thailand - success story (25% middle-aged men have stopped smoking)
  – Packet warnings very clear
  – Bans on tobacco advertising
  – Anti-smoking media campaigns
  – Positive efforts of high-ranking politicians
  – Lucky as both govt and NGOs active
Nicotiana tabacum
Forget for a moment that this plant is blamed for millions of deaths yearly

- Phillippine-
  - Tobacco will be turned in to
    - Food supplements
    - Antibiotic ointments
    - Skin creams
    - Building materials
    - Paints
    - Pesticides
    - Paper
  - Likely to supplement the tobacco farming sector
Forget for a moment that this plant is blamed for millions of deaths yearly

- Leaves – people smoke
- Seeds, stalks and roots – can be used for alternative harmless purposes
- Stems- are woody; good for particle boards and dissolving pulps
- India, US, UK experimenting with proteins from tobacco leaves
  - Nutritional supplement ??from poison
Nicotiana tabacum

Forget for a moment that this plant is blamed for millions of deaths yearly

- Tobacco was used for medicine before it was used for smoking
- Folk remedies formed basis for medicinal applications
- Antibacterial, antifungal creams and topical analgesics
- Minty smelling oil pressed from seeds
  - Can be turned in to soap and paint
- Whole seeds which are free from nicotine & high in animal protein
  - Potential as animal feed
Thank you for a patient hearing