

Occupational Cancer: A Global Perspective

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Presentation Outline

- **What do we know about occupational cancer and what is lacking?**
- **What are we doing now and what should we do in the future?**
- **Methodologic issues in occupational epidemiology**

What Do We Know About Occupational Cancer

- **A number of well established occupational carcinogens**
- **Many possible carcinogens with limited human data**
- **A number of occupations with elevated rates where the agent is unclear**
- **Many chemicals that cause cancer in animals with little human data**
- **Provided information on mechanisms of action**

Brief History of Occupational Cancer

- **1600s – Lung disease (cancer) in miners (Agricola)**
- **1713 – Diseases of workers (Ramazzini)**
- **1775 - Chimney sweeps and scrotal cancer (Percivall Pott)**
- **1875 - Coal tar and paraffin workers and scrotal cancer (many investigators)**
- **Early 20th century - High risk occupations identified, particularly for respiratory cancer**
- **Many hazards identified in 1950s-1980s**

Some Well-Established Occupational Exposures Causing Cancer

<u>Cancer Site</u>	<u>Exposure</u>	<u>Cancer Site</u>	<u>Exposure</u>
Bladder	benzidine	Mesothelioma	asbestos
	coal tars, TCE	Bone	radium
	naphthylamine	Larynx	acid mists
	aminobiphenyl	Liver	arsenic, TCE
Kidney	TCE		vinyl chloride
Lung	arsenic, asbestos, beryllium, chromium, chloromethyl ether, coal tars, diesel, radon silica, mustard gas	Nasal cavity	nickel, wood dust, chromium
		Skin	arsenic, coal tars, mineral oils, solar
Nasopharynx	formaldehyde	Leukemia	benzene, butadiene, formaldehyde,
All sites	Dioxin (TCDD)		ethylene oxide

What Conclusions Can We Draw from Established Carcinogens?

- Various cancers affected (respiratory predominates, but also urinary, blood, digestive, and bone)
- Digestive cancers less frequent
- Carcinogens are in many chemical groups
- Inhalation predominant route of exposure, but other routes not well investigated
- Not sure this chart tells us where to look next

Some Exposures Requiring Further Evaluation (IARC 1, 2A, 2B categories)

<u>Substance</u>	<u>Cancer</u>	<u>Substance</u>	<u>Cancer</u>
<u>Asbestos</u>	Gastrointestinal Kidney Larynx	<u>Butadiene</u>	Lymphoma
<u>Cadmium</u>	Prostate	<u>Styrene</u>	Leukemia
<u>Cutting oils</u>	Lung Skin	<u>2,4-D, DDT</u>	NHL
<u>Formaldehyde</u>	Nasal sinuses	<u>Lead</u>	Brain, Kidney
<u>Silica</u>	Stomach	<u>Diesel fumes</u>	Bladder Stomach
<u>Talc</u>	Ovary, Liver Brain	<u>Dust</u>	Lung
<u>Vinyl chloride</u>	Ovary Brain	<u>Pesticides</u>	Lymphoma, Leukemia, Myeloma, Lung, Prostate
<u>Shiftwork</u>	Breast, Ovary, Prostate	<u>Tetrachloroethylene</u>	Kidney, Liver Ovary, NHL

Occupations With Cancer Excesses Where The Agent Has Not Been Clearly Identified

<u>Occup Group</u>	<u>Cancer Site</u>	<u>Occup Group</u>	<u>Cancer Site</u>
<u>Farmers</u>	Leukemia NHL Lung Prostate Lip Stomach Brain Myeloma	<u>Dry cleaners</u>	Bladder Esophagus Kidney Liver Cervix
<u>Chemists</u>	Several Sites	<u>Petrochemical workers</u>	Leukemia Brain Kidney NHL
<u>Pattern makers</u>	Colon	<u>Rubber workers</u>	Leukemia Lung
<u>Welders</u>	Lung	<u>Meat workers</u>	Lung, Leukemia
<u>Hairdressers</u>	Leukemia		
<u>Waiters</u>	Lung		

Examples of Chemicals Causing Cancer in Animals Without Adequate Epidemiologic Data (From IARC, Supplement 7)

Chlordecone

Chloro-ortho-toluidine

Dichloroethane

Ethylhexyl phthalate

Diethylhydrazine

Ethyl acrylate

Methylene dianiline

Mirex

Nitropropane

Potassium bromate

Safrole

Styrene oxide

Sulfallate

Thioacetamide

Toluene diisocyanate

Vinyl bromide

There are many more.

Occupational Contributions to the IARC Classification of Carcinogenicity

Through 2004, IARC reviewed 880 factors for carcinogenicity

- **90 classified as human carcinogens**
 - 29 occupational (32%)
- **64 classified as probable human carcinogens**
 - 26 occupational (41%)
- **264 classified as possible human carcinogens**
 - 110 occupational (42%)

What Has Occupational Cancer Research Contributed to Our Understanding of Cancer Etiology

- **Identified many human carcinogens**
- **Accounted for a sizable proportion of the substances classified as human carcinogens by IARC**
- **Provided many leads that require further evaluation**
- **Made major contributions to our understanding of the carcinogenic process**

Issues Regarding Occupational Exposures

- **Lead to high cancer rates for a small proportion of the population**
- **Largely involuntary, e.g., not under control of individual workers**
- **Should have a high priority for intervention to prevent cancer occurrence**
- **All countries have some procedures to control exposure**
- **Exposure control is a mixture of workplace initiative and government regulation**

Occupational Exposure Control

How successful are exposure reduction efforts?

- **Exposure studies demonstrate that intervention reduces exposure levels***
- **Literature on disease reduction from exposure intervention is sparse@**
- **Studies specifically designed to address this issue are rare**

*Symanski et al. 1988. OEM 55:310-316.

@Tomatis et al. 1997. Carcinogenesis 18:97-105.

Cancer Reduction Following Asbestos Exposure Control

- **Literature review is difficult because computer searches are inadequate**
- **Reviewed 1094 epidemiologic papers on asbestos**
- **Found 8 with information on cancer risks following asbestos exposure intervention**
- **Most did not mention the intervention in the title or in key words**
- **Required reading abstracts or full paper**

Hohenadel K et al. The effectiveness of asbestos-related interventions in reducing rates of lung cancer and mesothelioma. (In review.)

Results from Review of Cancer Reduction Following Asbestos Exposure Control

Results:

- 8 papers with information on lung cancer and 6 on mesothelioma after intervention
- 6 of 8 showed reduction in lung cancer
- 4 of 6 showed reduction in mesothelioma

Hohenadel K et al. (In review.)

Examples of Studies of Lung Cancer Risk Following Asbestos Exposure Control

SMRs Following Exp. Control in 1930s (<u>Newhouse, 1989</u>)	SMRs Following Asbestos Regulation in 1969 (<u>Harding, 2009</u>)	RRs Years Since Halt of Production (<u>Magnani, 2008</u>)
<1940 1.53	<1930 0.83	<3 0.38
1941-50 1.06	1940-49 0.80	3 to 15 1.00
1951-60 0.96	1950-59 0.73	15 to 30 0.70
1961-70 0.90	1960-69 0.55	>30 0.56
>1970 0.88	1970-79 0.52	
	1980-89 0.38	
	>1990 0.17	

From: Hohenadel K et al. (In review.)

Examples of Studies of Mesothelioma Risk Following Asbestos Exposure Control

SIRs Following Control Efforts in 1950s (Ulverstad, 2002)		SMR Following Asbestos Regulation in 1969 (Harding, 2009)		RR Years Since Halt of Production (Magnani, 2008)	
<1942-51	99.0	<1930-39	NA	<3	0.67
1952-59	35.3	1940-49	1.00	3 to 15	1.00
1960-69	27.9	1950-59	3.22	15 to 30	0.90
		1960-69	1.71	>30	0.65
		1970-79	0.76		
		1980-89	0.76		
		>1990	1.02		

From: Hohenadel K et al. (In review.)

Conclusions from Literature Review of Cancer Reduction Following Asbestos Exposure Control

Findings:

- **Cancer is reduced following exposure intervention, but results are complex**
- **Reduction similar for lung cancer and mesothelioma**

Recommendations:

- **Need reviews to evaluate the literature on other occupational carcinogens**
- **Need studies specifically designed to evaluate changes in cancer rates following exposure intervention**

Current State of Epidemiologic Research on Occupational Cancer

Concern: The overall effort is diminishing

- Fewer papers published now than in the past
- Fewer sessions at scientific meetings
- Greater difficulty in accessing occupational groups to study
- Diminished funding

Number of Epidemiology Papers on Occupational Cancer Published from 1991 to 2009 in Occupational Major Journals

<u>Year</u>	<u>Number</u>	<u>Year</u>	<u>Number</u>
1991	71	2001	81
1992	82	2002	63
1993	91	2003	84
1994	107	2004	81
1995	88	2005	75
1996	84	2006	71
1997	87	2007	55
1998	88	2008	37
1999	91	2009	32
2000	89		

From: Raj et al. Am J Industr Med (In press).

Trends in Occupational Cancer Studies Around the World

Survey of occupational cancer papers published between 1991 and 2009 (N=1467)

- **Steep decline in U.S. and Canada**
- **Modest decline in Europe**
- **Small decline in Great Britain**
- **Slight increase in Asia**

From: Raj et al., AJIM (In press)

Evidence Regarding a Decrease in Funding, Access to Workers, and Occupational Sessions at Scientific Meetings

- **No direct evidence on these points, but all seem likely**
- **Providing quantitative information on these points would be useful**
- **Survey of occupational epidemiologists regarding state of the field might be instructive**

Why the Decrease in Number of Papers on Occupational Cancer?

Some believe:

- **Occupation is not an important contributor to the cancer burden**
- **All important carcinogens already identified**
- **Most occupational exposures well controlled**
- **Does not contribute to our understanding of carcinogenic mechanisms in “omic” era**
- **Political factors – first step in regulation**

What evidence do we have on these points?

What Proportion of Cancer is Due to Occupational Exposures?

<u>Country/Author</u>	<u>%</u>
New Zealand ('t Mannelje et al, 2005)	3.1-5.4
Men	5.2-8.7
Women	0.7-1.7
China (Wang et al, 2012)	2.7
Great Britain (Rushton et al, 2012)	5.3
Men	5.7
Women	2.2
Finland (Nurminen et al, 2001)	
Men	13.8
Women	2.2

Proportion of Cancer Due to Occupational Exposures: Continued

<u>Country/Author</u>	<u>%</u>
United States (Steenland, 2003)	
Men	3.3-7.3
Women	0.8-1.0
Alberta, Canada (Health Service, 2010)	
Men	3.3-13.8
Women	0.1-2.2
United States (Doll and Peto, 1981)	4.0

Occupational Cancer Burden Estimates: Summary and Conclusions

- **Range for men: 3.3 – 13.8%**
- **Range for women: 0.1 – 2.2%**
- **Most estimates less than 5.0%**

- **Compare these attributable fractions for occupation with those for other risk factors**

Proportion of Cancer Due to Various Factors (from Doll and Peto, 1981)

<u>Factor</u>	<u>%</u>
Diet	35
Tobacco	30
Infections	10
Reproductive/sexual behavior	7
Occupation	4
Geophysical factors	3
Alcohol	3
Pollution	2
Medicines	1

Conclusions Regarding Occupational Exposures and Cancer Burden

- **Two categories of causal factors**
 - **Diet and tobacco**
 - **Everything else - occupation, infections, alcohol, pollution reproductive/sexual behavior, medicines**
- **Proportion due to occupational exposures similar to all other factors except diet and tobacco**
- **Tobacco estimate very well documented**
- **Diet less well documented**

Additional Points Regarding Occupational Exposures and Cancer Burden

- **Larger than 4% for blue-collar workers (perhaps as large as 25%)**
- **Varies by cancer site and some cancers more deadly than others**
- **Lung a major occupational site and is particularly lethal**
- **Occupational exposures not voluntary**
- **Many exposures readily preventable**

What Don't We Know About Occupational Carcinogens

- **Women and minorities seldom studied in the past (changing now)**
- **Workers from small businesses rarely included in cohorts**
- **Most previous studies from developed countries, but a growing number in developing countries**

Inclusion of Women in Occupational Studies

Survey of occupational cancer papers published between 1991 and 2009 (N=1467) that included results on women

- **38% of papers from 1991 to 1995**
- **62% of papers from 2006 to 2009**

From: Raj et al., AJIM (In press)

Methodologic Needs for Future Studies on Occupational Cancer

- **Include quantitative exposure assessment**
- **Collect of information on non-occupational risk factors**
- **Assess mechanisms of action and evaluate effect modification from genes, lifestyle factors, and other occupational exposures**
- **More frequent use of cross-sectional, case-control, and prospective designs than in the past**

Use Prospective Cohorts in Occupational Studies

- Not a common design in occupational studies
- Surprising because of the frequent use of the historical cohort, a similar design
- Allows updating of information, biologic specimen collection, and evaluation of new issues
- Many literature reviews consider **ONLY** findings from prospective cohorts
- A 3 month literature survey in 2013 found 34 epidemiology reviews that only considered results from prospective cohort studies (none were on occupational exposures)
- But a letter on formaldehyde included a meta-analysis based only on cohort studies

Study Designs of Occupational Cancer Studies

Survey of occupational cancer papers published between 1991 and 2009 (N=1467)

Historical cohort	61%
Case-control	29%
Nested case-control	6%
PMR	3%
Cross-sectional	0.2%

From: Raj et al., AJIM (In press)

Type of Exposure Assessment in Occupational Studies of Cancer

<u>Type of Exposure Assessment</u>	Number of <u>Studies</u>	<u>%</u>
Occupation or Industry only	23	32
Occupation/Industry and duration	19	26
Ever/never for specific exposures	7	10
Qualitative estimates	15	21
Quantitative estimates	<u>8</u>	<u>11</u>
Total	72	100

From articles on occupational cancer published in the Scand. J. Work Environ. Health and the Amer. J. Industr. Med. over a two year period from 2002-04.

Number of Occupational Cancer Articles with Exposure-response Information

Survey of 1467 occupational cancer papers published between 1991 and 2009

1991-1995	27%
1996-2000	37%
2001-2005	42%
2005-2009	49%

From: Raj et al., AJIM (In press)

Promise of Molecular Epidemiology

- **Biologic markers of exposure**
 - Closer approximation of dose
 - Balances all routes of exposure
- **Biomarkers as measures of early outcomes**
 - Enzymatic effects
 - Changes in cell populations
 - DNA effects
 - Epigenetic changes
- **Gene-exposure interactions**
- **Better characterization of cancer types**

What Future Occupational Studies Might Look Like

Non-Hodgkin lymphoma as an example

Characteristics of Non-Hodgkin Lymphoma Pertinent to Study Design

- **Complicated pathology; many histologic types**
- **Clear relationship with immune system alterations**
- **Involvement of chromosomal translocations (e.g., t8;14 and t14;18, among others)**
- **Role for many environmental factors including solvents, pesticides, hair dyes, PCBs, PAHs, and viruses**
- **Role for genetic polymorphisms, particularly related to immune system, i.e., Th1, Th2 (cytokine genes) and TNF (tumor necrosis factor)**
- **Role for epigenetic mechanisms e.g., telomere length, methylation**

Occupation and NHL: Study Design

- **Collect biologic tissues to:**
 - **Characterize NHL subtypes**
 - **Evaluate immune genes and occupational exposures**
 - **Evaluate metabolic genes and occupational exposures**
- **Develop quantitative estimates of occupational exposures**
- **Obtain information on lifestyle factors**
- **Assess risk by NHL subtypes**

NHL Study in New Zealand

Study: Pesticide exposures and early biomarkers of NHL in farmers

Investigators: Andrea 't Mannelje and team

➤ **Exposure based on urinary measurements and information from questionnaires**

➤ **Biomarkers for:**

- **DNA damage, t14;t18 and q32;q21 translocations**
- **Telomere length**
- **Lymphocyte subsets**
- **Serum immunomarkers**

Methodologic Issues in Occupational Epidemiology

➤ **Confounding**

➤ **Exposure Misclassification**

(These are actually relevant for all epidemiologic studies)

Control for Smoking Confounding in a Case-Control Study of Lung Cancer and Occupation

<u>Occupational Category</u>	<u>Unadjusted OR</u>	<u>Smoking/Age Adjusted OR</u>
Professionals/technicians	0.9	1.1
Office/related personnel	1.0	1.1
Agric/forestry/fishery workers	1.4	1.5
Metal smelting and treatment	1.2	1.1
Chemical workers	1.6	1.4
Textile workers	0.7	0.7
Food/beverage workers	0.9	1.0
Printers	1.2	1.5
Pipe fitters/welders	0.9	0.9
Painters	1.6	1.4
Transportation equipment	1.1	1.1
Construction workers	1.6	1.4

From: Levin et al. Br J Ind Med 1988;450-458.

Control for Smoking and Asbestos Confounding in a Case-Control Study of Lung Cancer and Occupation

<u>Industry</u>	<u>Age Adj OR</u>	<u>Age/Smk Adj OR</u>	<u>Age/Smk/ Asbestos Adj OR</u>
Agric/forestry/fishing	1.3	1.3	1.3
Energy/mining	1.7	1.5	1.4
Chemical/oil	1.2	1.2	1.2
Stone/glass/pottery	1.8	1.6	1.5
Metal production	1.4	1.4	1.3
Electrical/sheet metal	0.9	0.9	0.9
Leather/textile	1.0	1.0	1.0
Construction	1.6	1.4	1.3
Financing/insurance	0.8	0.8	0.8
Restaurants/hotels	1.4	1.0	1.1

From: Bruske-Hohlfeld et al. Am J Epid 2000;151:384-395.

Relative Risks (# Exposed Deaths) for Lung Cancer by Cumulative Exposure to Acrylonitrile with Control for Smoking

Quintile of Estimated Exposure

<u>Analysis Group</u>	<u>Lowest</u>	<u>2nd</u>	<u>3rd</u>	<u>4th</u>	<u>Highest</u>
% Ever Smoked Cigarettes	62%	64%	68%	72%	75%
Entire Cohort	1.1 (27)	1.3 (26)	1.2 (28)	1.0 (27)	1.5 (26)
Entire Smoking Subcohort (Not Adj. for Smoking)	0.8 (27)	1.1 (26)	1.0 (28)	0.9 (27)	1.5 (26)
Smoking Subcohort with Smoking Data (Not Adj.)	0.3 (5)	0.9 (6)	1.0 (7)	1.0 (13)	1.7 (9)
Smoking Subcohort Adj. for Ever Used Cigarettes	0.3 (5)	0.8 (6)	1.0 (7)	0.9 (13)	1.6 (9)

From: Blair et al. Scand J Work Environ Health 1998;24:suppl 2:25-41.

Summary of Comparisons of Unadjusted RRs and RRs Adjusted for Various Factors from Papers in Six Issues of the Am J Epid

- **Four of 92 comparisons differed by >0.3**
- **Four of 92 might result in a different conclusion using adjusted RR**
 - **Two with a change in magnitude**
 - **Two with a change to no effect**

Confounding in Epidemiology

➤ **Conclusions:**

- **Confounding is rare – about 5% occurrence in a sample of papers from the literature**
- **Even when it occurs it seldom has a major impact**
- **Message: Should not suggest/assume confounding without some evidence that it occurs**

➤ **What if you cannot adjust directly?**

- **Are requirements for confounding evident?**
- **Are other effects of confounding apparent?**
- **Has this confounding occurred in other studies?**
- **Estimate possible effect (Axelson method for smoking)**

Misclassification of Exposure in Occupational Epidemiologic Studies

A concern in epidemiology because actual delivered dose almost never available

- **Biologic measures extremely rare**
- **Air measurements clustered in recent years**
- **Quantitative estimates desirable, but fraught with error**

Types of Misclassification of Exposure

- **Nondifferential** – misclassification same in cases as in noncases. Biases RR toward the null.
- **Differential** – misclassification not the same in cases and noncases. Can bias RR toward or away from the null depending on the orientation of the error.

Example of Typical Effects of Non-differential Exposure Misclassification

True Exposure Classification

	Yes	No	
Case	150	350	500
Control	50	450	500
	200	800	

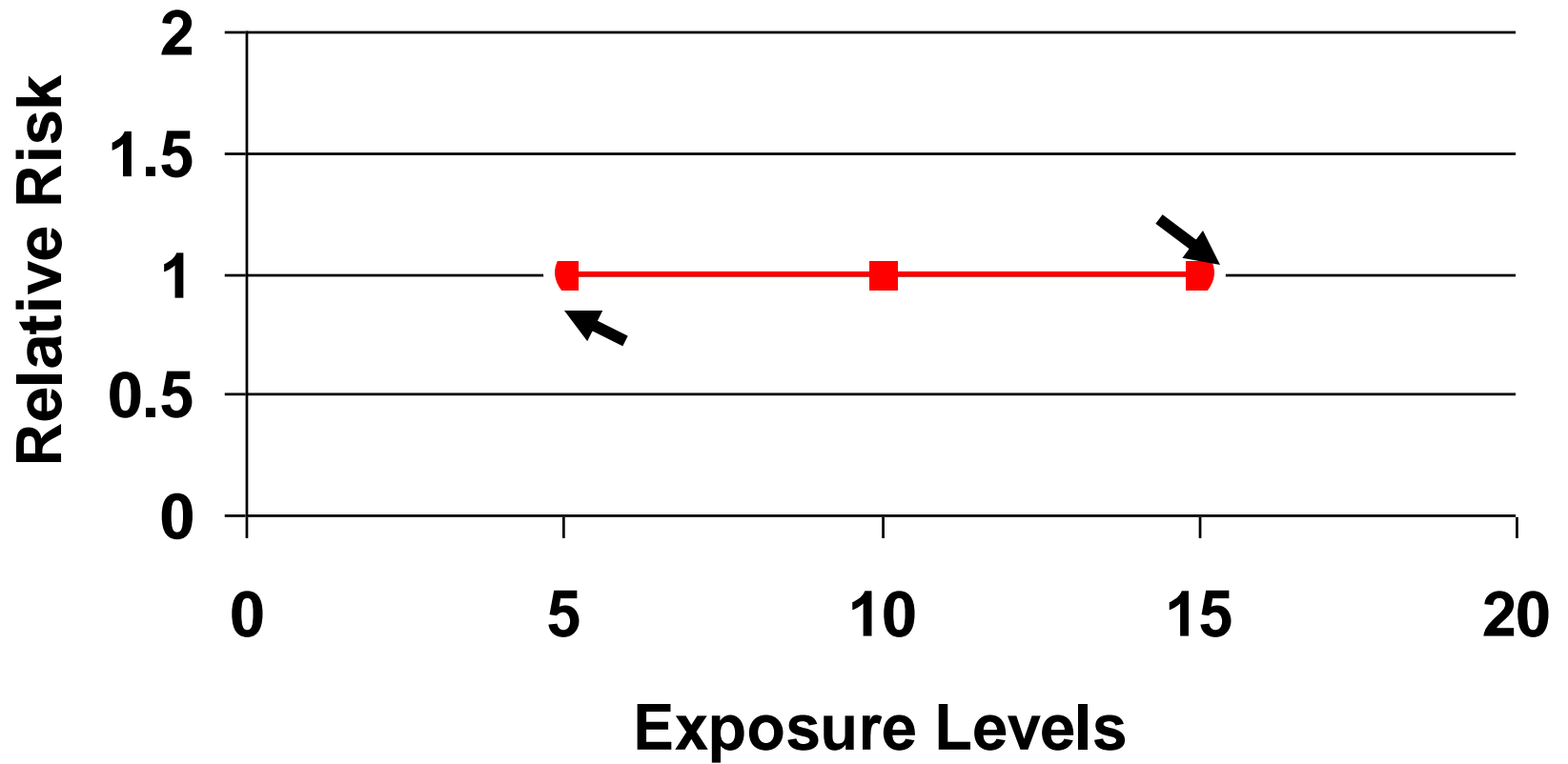
With 20% Non-differential Misclassification of Exposure

	Yes	No	
Case	190	310	500
Control	130	370	500
	320	680	

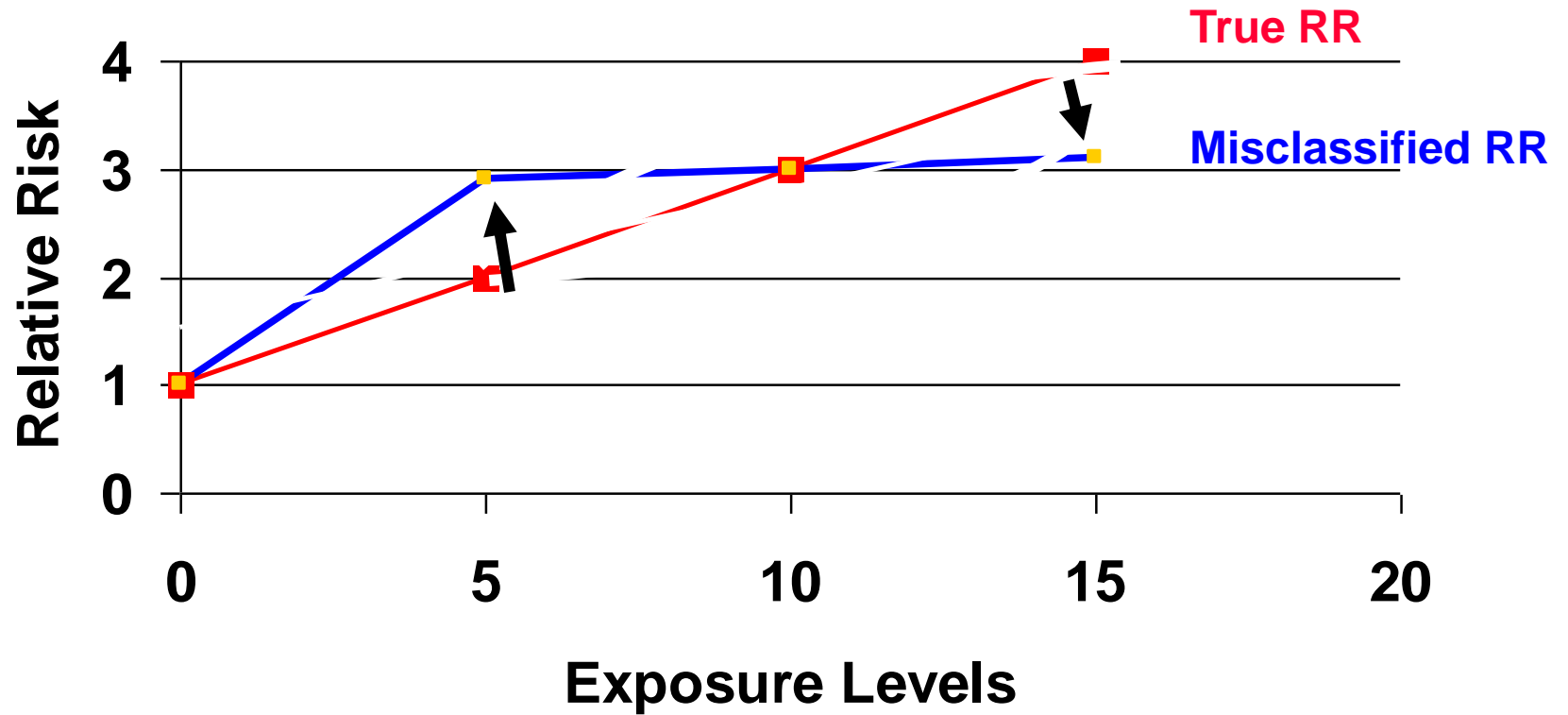
OR=3.9 — — — ► **OR=1.7**

In this example, the observed OR is attenuated by 56% when 20% of exposed cases (n=30) and controls (n=10) are misclassified as non-exposed, and 20% of non-exposed cases (n=70) and controls (n=90) are misclassified as exposed.

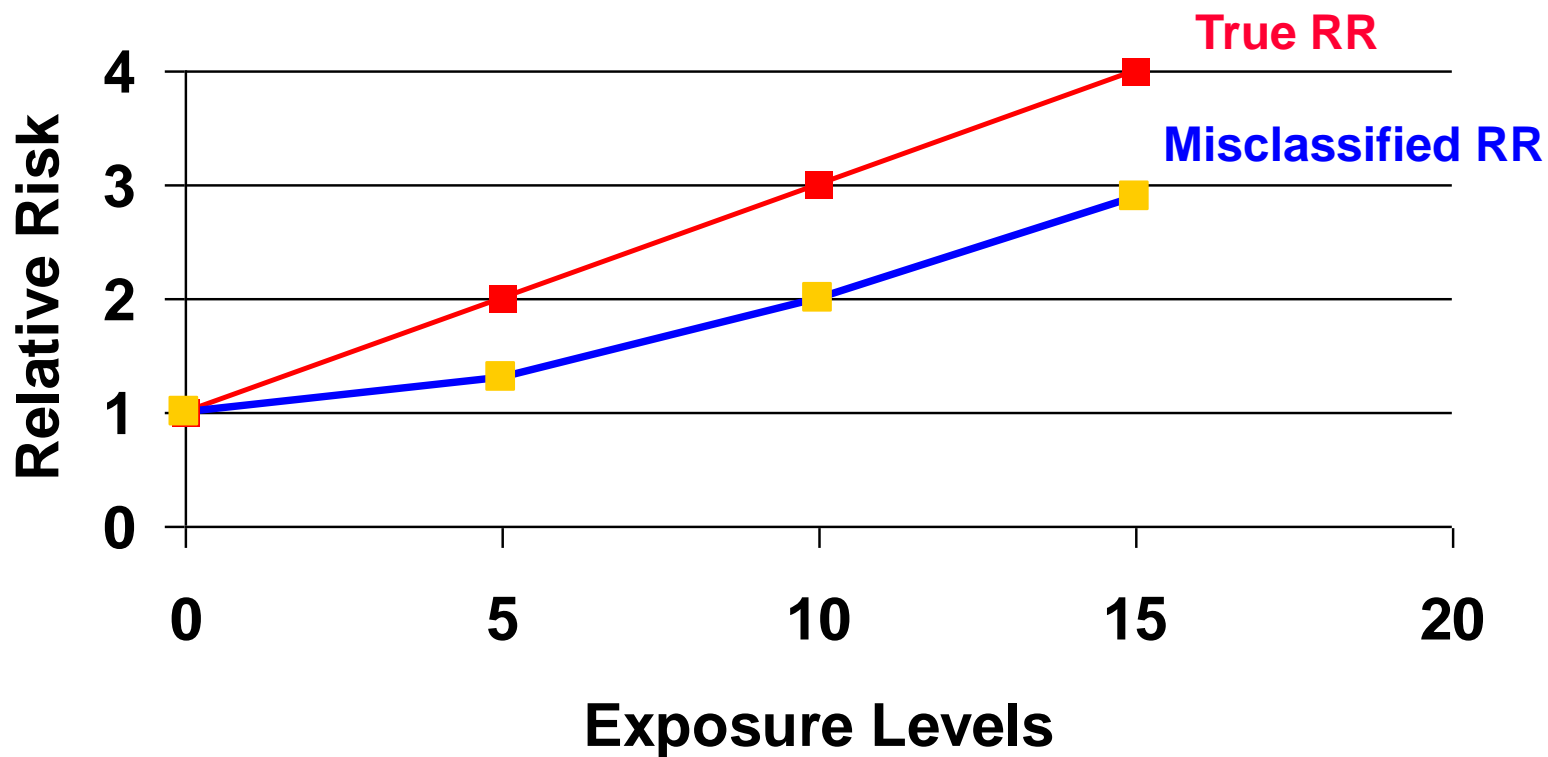
**Misclassification of Exposure:
True Association**
with nondifferential misclassification



Misclassification of Exposure: True Association



Misclassification of Exposure: True Association



Indication of Possible Levels of Misclassification in Occupational Studies

- Acrylonitrile – Measurements/estimates, $r = 0.6$
- Dioxin – Serum levels/estimates, $r = 0.70$
- Coal tar volatiles – Measurements/estimates, $r = 0.42$
- Formaldehyde – Different estimates, $r = -0.1$ to 0.7
- Jobs – Reported/recorded jobs, 83% agreement
- Welding fumes- Measurements/experts, $r = 0.42$
- Asbestos – Supplementary Qx/JEM, Kappa = 0.39
- 2,4-D – PK Model/urinary measurements, $r = 0.65$

Observed Relative Risks Based on Sensitivity, Specificity, Exposure Prevalence and True Risks

Sensitivity=0.7

<u>True RR and Exposure Prev.</u>	<u>Specificity=1.0</u>	<u>Specific=0.7</u>
True RR = 2.0		
Exposure prev.=10%	1.94 (0.81)	1.15 (0.19)
= 30%	1.80 (0.76)	1.30 (0.36)
= 50%	1.63 (0.70)	1.31 (0.40)
True RR = 3.0		
Exposure prev.= 10%	2.82 (0.81)	1.29 (0.19)
= 30%	2.44 (0.76)	1.53 (0.36)
= 50%	2.05 (0.70)	1.50 (0.40)

() = Kappas for corresponding sensitivity, specificity and exposure prevalence.

Misclassification of Exposure

- **Misclassification is the major weakness in ALL epidemiologic studies**
 - Not well considered in data interpretation
 - Ignoring it creates false negative impressions
- **To consider impact of misclassification**
 - Evaluate the likely degree of misclassification
 - Scour literature for relevant data and examples
 - Perform sensitivity analyses to estimate effects
 - Assess magnitude of misclassification in relation to other study biases and problems

Occupational Cancer

Contribution of genes and exposures

Sources of Information on the Contribution of Genes and the Environment on Cancer Development

Genes

- Family history increases risk
- Concordance among twins
- Role of specific genes

Environment

- Estimates of environmental contribution
- Changes in rates over time
- Immigrants assume risk of new location
- Risk factor studies

Heritable Portion of Selected Cancers

<u>Cancer</u>	<u>Heritable Proportion</u>	<u>Environmental Proportion</u>
Stomach	0.28	0.62
Colorectum	0.35	0.60
Pancreas	0.36	0.64
Lung	0.26	0.64
Breast	0.27	0.67
Cervix	0.0	0.80
Ovary	0.22	0.78
Prostate	0.42	0.58
Bladder	0.31	0.69
Leukemia	0.21	0.66

From: Lichtenstein P. et al. Environmental and heritable factors in the causation of cancer. New England J Med 343:78-85, 2000.

Cancer – Nature, Nurture, or Both

Conclusions by Robert Hoover, M.D.*

- Cancer is largely environmental (65 to 80%), **but** –
- Heritable component is not inconsequential (20 to 40%)
- Recent successes in molecular genetics have overshadowed studies of environmental factors
- Increase in knowledge in one area will benefit the other
 - Information on exposures should help identify genes
 - Study of genes related to cancer may help identify previously unrecognized environmental factors
- Risks from gene – exposure interactions can be reduced by removal of either

Summary Regarding Genes and Environmental Exposures

- **Cancer is largely of environmental origin**
- **BUT** genetic contributions are important
- **Many, many, many environmental and occupational leads**
- **Incorporate genetic components into traditional epidemiologic to strengthen studies and enhance understanding of causes and mechanisms**

Conclusions Regarding Occupational Cancer

- **Occupational cancer epidemiology has uncovered many of the established human carcinogens**
- **Many leads exist that require evaluation**
- **Both genes and exposures important**
- **Studies integrating epidemiology, toxicology, and genetics offer the best opportunities**
- **High-quality exposure assessment essential**
- **Confounding must always be considered, but is seldom a critical problem**

Occupational Cancer: Future Needs

- **Continued research to identify and clarify cancer hazards in the workplace**
- **Proactive intervention efforts to control exposure**
- **Investigations to evaluate the effectiveness of intervention for exposure control and disease reduction**
- **Development of a fuller understanding of cancer risks from occupational and environmental exposures**

Cancer Prevention and Control Lecture

Occupational Causes of Cancer

Questions?