

# Occupational COPD

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May 13th, 2016

# A small case...

- 77 y. old man, referred for occup. COPD
  - 19-29 : construction & shops
  - 29-58: foundry
    - Maintenance of road tracks inside the building
    - Regular small fire with spills of molten metal, that he extinguishes
    - Often sooty at end of shift; dusty environment...; no RPE

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- **Smoking history** – very variable
  - 22-65 y : 12-15 cig./d, stopped 2 mo. ago (HbCO 2.9%) – 22.5 p-y
  - Seen by a chest physician a year ago: 50-55 p-y, still active
- Cough & sputum for many years
- Dyspnea for >20 y. and progressive; 3-4/5
- Taking regular long-acting bronchodilators
- FEV<sub>1</sub>/FVC : 1.07/1.75 → 0.98/1.75
- CPT 103%; VC 60%; FRC 128%; RV 158%
- **DLCO 35%**; 7.42, 43, 55

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- Conclusion of WCB (Qc)
  - No occupational lung disease
  - COPD with emphysema, **smoking-related**
  - Based on high cigarette consumption and perceived medium exposure to dust and fumes (fire)

# Criteria used in Quebec by WCB

- Agreed relationship to work if
  - < 20 p-y
  - and
  - > 20 y. ex exposure to high level of VGDF
- Are we right to make this conclusion ?

# How do we determine causality of COPD?

- It's one thing to state that epidemiologic studies confirm higher risk of COPD in various occupational settings, it is quite different to state that work exposure to VGDF is responsible for the development of COPD in an **individual** worker.
- Cigarette smoke (and pollution in underdeveloped countries) is the major cause of COPD, even in workers exposed to VGDF
- Cigarette smoke has at least an additive effect to VGDF

# Determining causality...

- On individual basis, we must
  - Determine whether **exposure** to VGDF is sufficient to induce COPD
  - Take into account
    - **Smoking** history
    - Other host factors (alpha-1 antitrypsin deficiency)
- However...
  - How can we objectively assess VGDF exposure ?
  - Is history from workers reliable in terms of respirable dust?

# Occupational COPD in the world

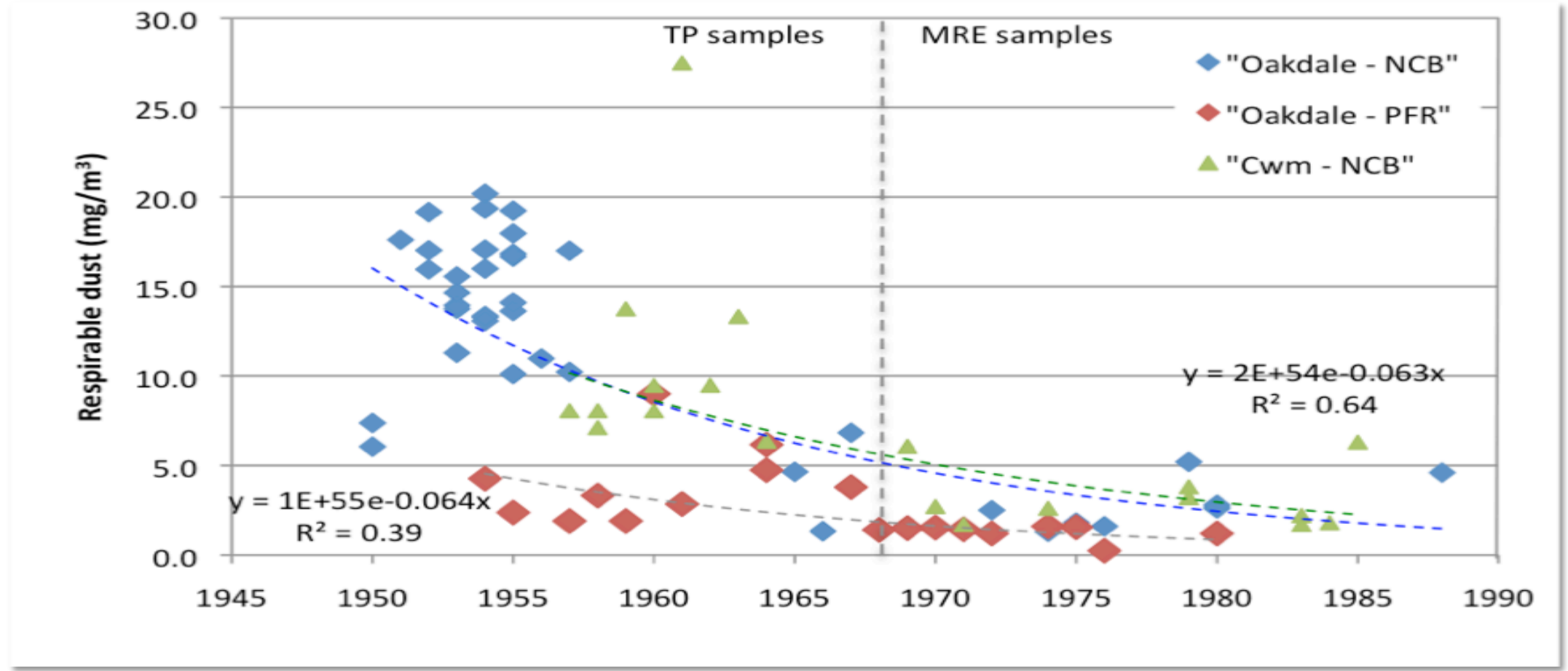
- In **Canada**, WCB have recognized only a few cases of Occup. COPD
  - Assessed on a case-by-case basis based on medical « experts » or committees
- In **Korea**, it is limited to workers exposed to high concentrations of coal mine dust, silica, or cadmium fumes for prolonged periods - Park SY, 2014
- In **France**, accepted if listed in tables (coal & iron mines, cotton, Ar), FEV1<40%P & sufficient duration of exposure or if recognized by a committee of 3 medical experts (when exposures are not listed in the tables & if disability is >25% or death) - Andujar P, 2016
- In **UK**, recognized if >20 y exposure of coal face work or cadmium fume exposure



# What is sufficient and relevant exposure ?

- Criteria vary between countries
  - Most require at least 20 years of exposure to a recognized agent (coal, cadmium)... less in France
  - There is no specified level of exposure ... and exposure has varied over time...

# Exposure to dust has decreased over time...



## Smoking as a confounder ...

- COPD is seen in never smokers, particularly those exposed to indoor pollution, but also to VGDF.
- Smoking is surely the most important factor in the development of COPD
- However, many jurisdictions look at all the factors and asks if it is **more likely than not that work made a significant contribution to the development of the disease** (as opposed to if work was the predominant contribution to the development of the disease)
  - If it is determined that work made a significant contribution to the disease, then the claim is allowed.

# Imputation – smoking vs work ?

## WSIB report – 2000 – review of literature

- Average loss of FEV1 with age : 33.6 ml/y
- Average loss of FEV1 with smoking : 8.5 ml/pack-years
- Average loss of FEV1 with dust: 3.0 ml/mg/m<sup>3</sup>-year

## Semi-quantitative estimation of the relative effects of dust and tobacco smoke

- Consider a worker exposed for 20 years to respirable dust at a level of about  $2 \text{ mg/m}^3$ 
  - an average worker can expect to see a FEV1 loss of about 120 mL ( **$2 \text{ mg/m}^3 \times 3.0 \text{ mL/mg/m}^3 \times 20 \text{ years}$** ) above that seen with normal ageing.
  - a 20 year pack per day cigarette habit would result in an average 170 mL ( **$20 \times 8.5 \text{ ml/pack-yrs}$** ) loss in FEV1
  - **Total loss due to dust:  $120/120+170 = 41\%$**

**TABLE III: PERCENTAGE CONTRIBUTION OF DUST EXPOSURE TO LOSS OF FEV<sub>1</sub> IN CLAIMANTS WITH COLD**

Dust Exposure (@ 2 mg/m <sup>3</sup> resp.), in years	Smoking, in pack-years																
	0	5	10	15	20	25	30	35	40	45	50	55	60	65	70	75	80
20	100	73	58	48	41	35	31	28	25	23	21	20	19	17	16	15	15
25	100	77	63	53	46	41	36	33	30	27	25	24	22	21	20	19	18
30	100	80	67	58	51	45	41	37	34	31	29	27	25	24	23	21	20
35	100	83	70	61	54	49	44	41	37	35	32	30	28	27	25	24	23
40	100	85	73	65	58	52	48	44	41	38	35	33	31	30	28	27	25
45	100	86	75	67	61	55	51	47	43	41	38	36	34	32	30	29	28
50	100	87	77	69	63	58	53	49	46	43	41	38	36	34	33	31	30

# Estimation of the contribution of smoking vs work in COPD by physicians

- Although there is no established criteria, Fishwick et al (2013) found that:
  - Although there was variability in the relative attribution assigned, expert physicians (COPD and Occupational Physicians) appeared to be assessing the relative occupational contribution, in part, as an inverse function of the contribution of smoking.
  - 20 p-y was similar to 20 y. of high-risk exposure
  - COPD experts assigned higher work-related contribution to COPD

# Disability

- A worker may be totally disabled but his impairment due to work may be less, taking into account smoking



# In conclusion...

- It is possible to establish the role of work exposure to VGDF in the development of COPD but there is generally no well established criteria and the majority of jurisdictions rely on experts and list of recognized agents.
- Is there a minimum impairment required to accept a claim?
- How can exposure be adequately assessed ?
- Is a history of heavy smoking enough to exclude occupational COPD?