COPD and environmental risk factors other than smoking

4. Alcohol

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1. Papers identified

Using the procedures described in "COPD and risk factors other than smoking. 1. Identifying Relevant Papers", 39 papers were identified as relevant.[1-39] One[17] provides corrected results to an earlier publication.[15]

2. Specific studies

25 of the papers describe the results of 24 studies relating alcohol consumption to COPD or an index related to it. In only about half of these is alcohol a major interest of the paper as judged by a reference to alcohol in the title of the paper. The rest are more general studies, with alcohol one of a range of risk factors studied. Table 1 summarizes the findings from these studies.

The results do not consistently show a beneficial or adverse effect of alcohol consumption. The studies can be divided into groups:

(a) Beneficial effect Three studies[23,25,35] report a beneficial effect of alcohol. One[25] is an autopsy study of emphysema, and another study[35] is a very large study.

(b) J-shaped curve Two studies[34,36] have reported results that seem to fit in with a J-shaped curve, somewhat like the relationship of IHD to alcohol, with an apparent beneficial effect for moderate alcohol consumption.

(c) No significant relationship Seven studies[2,4,5,11,16,30,32] report no significant association of alcohol consumption with the endpoints studied.

(d) Adverse effect Nine studies[12,14,15,18,19,22,27,33,39] reported an adverse effect of alcohol consumption, virtually always in analyses

adjusted for smoking. In some of these studies the effect seemed limited to heavier drinkers.

(e) Mixed findings Three studies[6,13,31] report somewhat conflicting results, as indicated in Table 1.

Overall these data do not appear to demonstrate convincingly whether alcohol consumption increases or decreases risk of COPD, but a more detailed overview would be needed to clarify the picture further.

2.2 Studies in alcoholics

11 of the papers describe studies of alcoholics. Table 2 summarizes results from these studies. It is noticeable that the papers are all quite old, with six studies published before 1980 and the latest only published in 1991. Given the strong relationship between heavy drinking and heavy smoking, it would seem essential to take account of smoking in analysis, but only six of the 10 studies at least attempted to do so. Of those that did, two of the studies[7,8] thought that the increased lung pathology in alcoholics could not be explained by their smoking, while two[20,26] thought that it could be. One study[9] reported an adverse effect of alcohol on COPD in light smokers but a beneficial effect in heavier smokers, while a study in nonsmoking alcoholics[28] found no effect of alcohol on lung function. All these studies are quite small. While the largest study[29] did not consider smoking, it was of some interest in that deaths from COPD were quite rare in alcoholics. Overall these data do not appear to demonstrate any clear effect of excessive alcohol consumption on COPD.

3. Reviews

The publications include three reviews[3,10,37]. The first,[3] published 40 years ago, "Alcoholic lung disease – an hypothesis," is more of a suggestion that "the frequent association of alcoholism and pulmonary disease is due, at least in part, to direct damage of lung tissue by alcohol."

The second,[10] "Alcohol and the Lung. A brief review," concludes that "Prolonged alcohol abuse affects the ventilatory functions of the lung …,

primarily by causing airway obstruction and diffusion limitation." However, the evidence considered is mainly non-epidemiological and there is mention of only one study that adjusted for smoking and that study[7] is very small.

The third review,[37] "The epidemiology of COPD," is wide-ranging, but contains a page and a half on alcohol. The authors note that the studies "have neither been as extensive as the smoking studies, nor have the findings been consistent in relation to the occurrence of airways obstruction or chronic bronchitis."

A number of the "Step 1 reviews" described in my first report also considered alcohol. Generally these conclude that the evidence is variable and inconclusive.[40-42]

4. Conclusion

It may be that heavy alcohol consumption has a small effect on the risk of COPD, but this is not well demonstrated.

5. References

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TABLE 1 Summary of general population studies of COPD and alcohol consumption

Source	Study design[a]	Alcohol index	Endpoint[b]	Result	Adjusted for smoking
Boutin-Forzano et al (2007)[2]	C	Occasional or regular vs never or former	CBE	No association	No
Chen et al (2000)[4]	C	Regular drinking	COPD	"No discernible association"	No
Cohen et al (1980)[5]	C	Heavy, moderate or light	AO, AR	No association	Yes
Dean et al (1977)[6]	CC	Heavy, moderate or light; Type of drink	COPD	Increased for heavy (males) Increased for beer (males), decreased for wine (males)	Yes
Higgins et al (1982)[11]	P	Ex, heavy or moderate, light or none	OAD	No significant association	No
Hrubec et al (1973)[12]	T	Ever intoxicated	CB	Increased for ever intoxicated	Yes
Kauffmann et al (1993)[13]	C	>30, 1-30, 0g per day	FEV_1	Decreased with increasing alcohol in Lewis-positive, increased with increasing alcohol in Lewis-negative	Yes
Lange et al (1988)[14]	P	5 levels of weekly alcohol	FEV_1 and FVC change	Increased loss over time with increasing alcohol	Yes
Lange et al (1990)[15,17]	P	Drinks/day	FEV_1 change	Increased loss over time in men, not women with increasing alcohol	Yes
Lange et al (1990)[16]	P	5+, 1-4, 0 drinks per day	OLD	No association	Yes
Lange et al (2003)[18]	C	3+ drinks per day	CB	Increased for 3+ drinks per day	Yes
Lebowitz (1981)[19]	C	Heqvy, moderate, none or light	FEV_1 and FVC	Decreased particularly in heavy smokers	Yes
Metzner et al (1983)[22]	C	5+ drinks a day	CB, FEV_1	Increased CB, reduced FEV_1 for 5+ drinks per day	No
Miedema et al (1993)[23]	P	Any alcohol	CNSLD	Reduced risk for drinkers	Yes
Pratt and Vollmer (1984)[25]	CC(A)	Heavy, slight or moderate, none	EMP	Prevalence and extent decreased with increasing alcohol	Yes

TABLE 1 (cont'd) Summary of general population studies of COPD and alcohol consumption

Source	Study design[a]	Alcohol index	Endpoint[b]	Result	Adjusted for smoking
Saric et al (1977)[27]	C	>1, >½-1, >0-½, 0 litres wine per day	CB, FEV_1<80%	Increase with increasing alcohol	Yes
Shin et al (2003)[30]	C	≥22, 8-21, 1-7, 0 drinks/wk	AO	No association	Yes
Sisson et al (2005)[31]	C	8 categories of drinks/month	OLD	Increased in former heavy drinkers	Yes
Sparrow et al (1983)[32]	P	Weekly consumption	FVC_1 and FVC	No association with level or change over 5 years	Yes
Ström et al (1996)[33]	C	Grams per day	EMP (TLC)	Increased with high consumption	Yes
Suadicani et al (2001)[34]	C	>35, 22-35, 1-21, 0 drinks/wk	CB	J-shaped relationship with lowest risk in 1-21 group	Yes
Tabak et al (2001)[35]	C	>30, 1-30, <1 g/day	FEV_1	Increased in drinkers	Yes
Tabak et al (2001)[36]	P	Grams per day	COPD	Decreased in light drinkers	Yes
			Lung function	Increased in light drinkers (J-shaped curve)	
Whicker et al (2006)[39]	C	Alcohol abuse	COPD	Increased in abusers	Yes

[a] Abbreviations used for study design: C = cross-sectional, CC = case-control, CC(A) = case-control (autopsy, study), P = prospective, T = twin.

[b] Abbreviations used for endpoints: AO = airway obstruction, AR = airway restriction, CB = chronic bronchitis, CBE = chronic bronchitis or emphysema, CNSLD = chronic non-specific lung disease, COPD = chronic obstructive pulmonary disease, EMP = emphysema, EMP (TLC) = emphysema (as measured by total lung capacity), FEV_1 = forced expiratory volume in one second, FVC = forced vital capacity, OAD = obstructive airway disease, OLD = obstructive lung disease.

TABLE 2 Summary of results from studies of alcoholics

Source	Population studied	Results	Smoking adjusted for
Banner (1973)[1]	30 alcoholics	"These data suggest that diffusion impairment and mild obstruction are characteristic of chronic alcoholism"	No
Emirgil et al (1974)[7]	23 alcoholics	91% had CB and 96% had abnormal lung function. "An attempt was made to separate the pulmonary effects of alcohol from (1) the effects of previous pulmonary infections, (2) the effects of cigarette smoking, and (3) the effects of cirrhosis of the liver. The data suggest that either alcohol itself through some unknown mechanism may be a causative agent in producing lung disease or that alcohol makes a higher percentage of the population susceptible to the harmful effects of cigarette smoking"	Yes
Emirgil et al (1977)[8]	44 former alcoholics	"This high incidence of abnormalities among both the men and women could not be attributed to previous pulmonary infection or smoking. Comparison of these patients with chronic alcoholics suggests that the obstructive component in these patients is, in part, a result of their past alcoholic intake and that it is not reversed by abstinence."	Yes
Garshick et al (1989)[9]	165 subjects (including 91 alcoholics)	"Lifetime alcohol consumption was also a predictor of lower levels of FEV_1 in a model that included age, pack-years of cigarette smoking, and an interaction between alcohol consumption and pack-years. The interaction between smoking and alcohol consumption was in a direction opposite to the independent effects of alcohol and smoking, suggesting a protective effect of alcohol with heavier amounts of smoking."	Yes
Lyons et al (1986)[20]	27 alcoholics	"The high prevalence of respiratory disease in alcoholics is largely attributable to their smoking habits: no evidence of a specific pulmonary toxic effect of ethanol was identified in the study."	Yes
Mellstrom et al (1981)[21]	54 recidivists[a] and 435 non-recidivists	Lifetime incidence of chronic bronchitis higher in recidivists	No
Pell et al (1968)[24]	922 problem drinkers and 922 matched controls	No significant differences in chronic bronchitis or emphysema	No
Rankin et al (1969)[26]	125 alcoholics	"The results have shown that chronic obstructive lung disease is the characteristic pulmonary illness of the alcoholic. As with the non-alcoholic, it is related to smoking habits and social class. The higher frequency of chronic bronchitis in alcoholics than in non-alcoholics can be explained by the former being twice as likely to be current smokers as the latter."	Yes
Sarkar and Gupta (1980)[28]	10 nonsmoking alcoholics	"Pulmonary function, including diffusing capacity, is within normal limits."	Yes
Schmid and de Lint (1969)[29]	6514 alcoholics	Of 738 deaths over a 14 year period, only 8 were of respiratory diseases (excluding pneumonia) and COPD was not mentioned as being increased	No

TABLE 2 Summary of results from studies of alcoholics (cont'd)

Source	Population studied	Results	Smoking adjusted for
Umbricht-Schneiter et al (1991)[38]	1524 patients screened, 28 with a principal discharge diagnosis and 117 with a secondary discharge diagnosis that was alcohol-related	Increased risk of COPD/asthma in both groups. Also seen in 295 subjects with a positive screen for alcohol, but no discharge diagnosis	No

[a] Recidivists attended two or more times at the Temperance Board.