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Weight change over eight years in relation to alcohol consumption in a cohort of continuing smokers and quitters

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Abstract

Aim

To examine the association of baseline alcohol consumption with weight change in smokers and quitters.

Design

Eight year prospective cohort study.

Setting

Oxfordshire general practices nicotine patch/placebo trial with 8 year follow-up.

Participants:

698 smokers attempting to stop smoking, of whom 85 were abstinent for eight years and 613 failed to stop smoking and smoked for eight years.

Measurements:

Weight was measured at baseline and at 8 years. Weekly alcohol consumption was reported at baseline. Regression models were used to examine weight change by baseline alcohol consumption in quitting and continuing smokers

Findings:

The effect of alcohol consumption on weight change depended upon smoking status (p for interaction=0.019). In smokers, alcohol had little association with weight change, 0.005[-0.037, 0.056]kg per UK unit/8g of ethanol consumed. This was unmodified by gender and baseline BMI. In quitters, the effect was -0.174[-0.315, -0.034]kg per unit (unmodified by gender and baseline BMI). Quitters who consumed 14units(112g ethanol)/week weighed a mean 2.4kg less after eight years than quitters who did not drink.

Conclusions

This evidence suggests a dose response relationship whereby quitters gain less weight per unit of alcohol consumed at baseline. This is consistent across studies, it may be accounted for by unmeasured confounders or it may be a real weight gain attenuating effect of alcohol. If a real effect, adverse health consequences of increasing alcohol need to be investigated against the benefit of smaller weight gain. Increasing alcohol should not currently be advised for preventing weight gain during smoking cessation.

Introduction

Eighty three percent of smokers that stop smoking gain weight, on average, 7kg more than if they had continued to smoke [Lycett et al, 2011]. Preventing this weight gain is important because it reduces the health benefits otherwise seen by quitting [Davey-Smith et al, 2005, Gerace et al, 1991, Chinn et al, 2005]. Avoidance of alcohol is often advocated in smoking cessation support to reduce cues to smoking. People trying to lose weight are often advised to moderate or avoid alcohol because alcohol has a combustible energy value of 7kcal/g. (Fat contains 9kcal/g and carbohydrate contains 4kcal/g). However there is contrary evidence on the effect of alcohol on weight gain as described below.

Laboratory studies of the metabolic effects of alcohol show three important effects on energy balance. Firstly, alcohol increases energy intake [Tremblay et al, 1995, Westerterp-Plantenga & Verwegen, 1999, Buemann, 2002], particularly when consumed in combination with fat [Tremblay & St-Pierre, 1996] and the extra energy from alcohol is not compensated for by reducing subsequent food intake [Tremblay et al ,1995, Tremblay & St-Pierre, 1996, De Catro & Orozco, 1990, Yeomans, 2004]. Secondly, alcohol suppresses fat oxidation, which increases fat storage [Suter et al, 1992]. Thirdly alcohol increases 24 hour energy expenditure through inducing thermogenesis by up to 30%. Dietary induced thermogenesis from alcohol is greater than that from carbohydrate, fat, or protein [Suter et al, 1994, Raben et al 2003, Schutz, 2000, Westerterp, 2004]. The first two effects point towards alcohol promoting weight gain and the last effect works against it. So what causes the balance to tip one way or the other?

The answer may depend on the pathway by which alcohol is metabolised. The alcohol dehydrogenase (ADH) pathway produces ATP more efficiently than the microsomal ethanol oxidizing system (MEOS). It is thought that low levels of alcohol are metabolised by ADH, whereas a high blood concentration of alcohol induces MEOS; and it is this that accounts for the high energy expenditure seen in people with alcohol dependence [Suter, 2005, Levine et al, 2000]. The threshold level of alcohol for MEOS induction is unknown, but individual variation of body weight, smoking status, gender, and genetic variation in enzymes metabolising alcohol may explain the different effects of alcohol on body weight [Suter, 2005].

For ethical reasons there are very few studies where increasing alcohol is used as an intervention. However those that have been done have found either no effect of alcohol on weight in obese subjects [Beulens et al, 2006] or a weight attenuating effect of alcohol which is more pronounced in lean rather than obese men and women. [Clevidence et al, 1995, Crouse & Grundy, 1984]

Cross sectional studies have shown inconsistent results, although this has been explained in part by gender effects, smoking status and drinking patterns. Lower alcohol intake has been associated with greater BMI in women, but less so, or with the opposite effect, in men [Hellerstedt et al, 1990, Golditz et al, 1991, Breslow & Smothers, 2005]. This effect in men is not consistently seen among male smokers [Mannisto et al, 1996, Cooke & Frost, 1982]. More frequent drinking has been associated with lower BMI independent of total alcohol consumption [Tolstrup et al 2005, Mannisto et al, 1996, Tolstrup et al, 2008].

Prospective studies have demonstrated increasing alcohol consumption over time is not associated with an increase in waist circumference [Tolstrup et al, 2008], but it is associated with weight gain [Gordon & Kannel, 1983], particularly in men [Gordon et al, 1986, Wannamethee & Shaper, 2003]. These studies have not investigated effect modification by smoking status. Prospective studies in women have found a U shaped curve [Wannamethee et al, 2004] and a significant inverse relationship on weight gain which was not modified by smoking status [Wang et al, 2010].

Three prospective studies (in males and females) have considered the effects of alcohol on body weight around the time of quitting smoking, these have all found an inverse effect of alcohol consumption and weight gain. In two studies the quitters were self reported as abstinent [Froom et al, 1999, Kawachi et al, 1996]. The third study investigated the effect in biochemically validated quitters, but did not compare this with the effects in continuing smokers[Nides et al 1994]. The weight gain trajectory in quitting and continuing smokers is very different; our paper is the first to investigate the effect of alcohol consumption on post cessation weight gain in both continuously abstinent quitters and continuing smokers.

Method

Participants

1686 participants, aged between 25 and 65 years, smoking 15 or more cigarettes a day had enrolled in a stop smoking clinical trial in 19 Oxfordshire general practices between June 1991 and March 1992 [ICRF, 1993]. They were randomised to either a 21mg nicotine patch or placebo for 12 weeks. They were instructed to quit smoking on trial entry and were seen

at 1, 4, 8 and 12 weeks. They were followed up at 6 and 12 months[Fowler, 1994] and 8 years[Yudkin et al 2003]

At 8 years 1625 participants were living, we were able to trace and contact 1532 participants. Of these 840 (52%) responded and provided us with data. Responders compared to non-responders were older (43.0 vs 41.5 years p=0.010), more likely to be female (59% v 52% P<0.005), and have stopped smoking during the trial (13% v 6% P<0.0001 quit for 1 year).

85 participants had been biochemically proven continuously abstinent from 3 months to 8 years (quitters), and 613 smoked continuously during this time (smokers). 116 smoked during the first year but quit at some point between years 1 and 8. The remaining 26 participants quit during the first year but relapsed by 8 years. Quitters gained 8.79kg, (SD 6.36), [95%CI 7.42, 10.17] over 8 years and continuous smokers gained 2.24kg (SD 6.65), [95%CI 1.70, 2.77][Lycett et al, 2011].

Ethical approval for this was granted by Anglia and Oxford Multicentre Research Ethics Committee, and 86 local research ethics committees[ICRF, 1993].

Measurements

Smoking Status

During the trial smoking abstinence was confirmed by expired CO<10ppm while participants were using the nicotine patch, and by salivary cotinine <20ng/ml after they had stopped

using the patch. Cotinine is considered a more reliable measure of abstinence but, as it is a metabolite of nicotine, it cannot confirm abstinence during nicotine replacement therapy.

At 8 year follow-up participants completed questionnaires on smoking history and quit attempts that spanned the last 8 years and smoking status was confirmed by salivary cotinine. Where no sample was supplied (9%) participants were considered to be smoking.

Weight Gain

Height and weight were measured at trial entry, although this was self-reported in 19% of participants. At eight year follow up, weight was self-reported as the questionnaire was completed by post. We found no significant differences between self reported and measured weight or BMI and this is discussed elsewhere [Lycett et al, 2011]. Change in weight was taken as the value of weight (kg) at 8 years minus the value of weight at baseline (kg).

Alcohol Consumption

Baseline data on weekly units of alcohol was collected by a trained nurse interviewer. Alcohol consumption was assessed by asking participants to report daily consumption of beer, lager or cider (pints), wine (glasses), sherry, vermouth or port (glasses), and spirits or liqueurs (single tots). Total weekly consumption was then converted to UK units (equivalent to 8g ethanol) per week. Alcohol consumption data was missing for 1 quitter and 17 smokers and these were excluded from the analysis.

Analysis

We analysed a sub-cohort of our sample (680 of 840) they were those who were continuously abstinent (84) and those who smoked continuously for the whole 8 years (596). This meant that recent or frequent changes of smoking status did not affect the results. These were all those smokers who achieved abstinence by the end of treatment and maintained this for eight years and all those smokers who attempted to stop smoking but were smoking at the end of treatment, at one year follow up, and at eight year follow up.

All statistical tests were carried out using Statistical Package for Social Sciences version 15 for windows software (SPSS 15.0). Linear regression analysis was used first with the combined cohort of continuous smokers and quitters to examine the effect of baseline alcohol consumption on weight change. We used higher order terms to investigate curvilinear relationships. We investigated effect modification by gender, baseline BMI and smoking status using these terms and the appropriate multiplicative interaction terms. As smoking status modified the effect of alcohol on weight change, separate regression equations were used for smokers and quitters. In both sets of equations confounding was controlled. Categorical variables (treatment allocation, gender, ethnic group, and socio-economic status measured by the Registrar General's classification [Drudy, 1991] were recoded into dummy variables. Continuous variables (BMI, height, age, number cigarettes/day, cigarette dependence measured by the Horn Russell (HR) score [Russell, 1974], and weekly alcohol consumption) were mean-centred. To avoid over-fitting, these potential confounders were entered in a stepwise selection process with a p value of 0.2 for model entry [Rothman & Greenland 1998]. Regression parameters and the 95% confidence intervals (CIs) are shown together with the p values and R squared statistics as appropriate.

We used Cook's distance as a measure of influential cases; if Cook's distance exceeded 1 we considered that outliers had the potential to influence our findings.

Results

Baseline characteristics

Baseline characteristics can be found in Table 1.

Baseline alcohol consumption as an effect modifier of weight change according to smoking status

In the model including smokers and quitters, weekly alcohol consumption at time of quit attempt was not associated with weight change. However, there was a significant interaction between smoking status and alcohol consumption before (p=0.019) and after (p=0.010) adjustment for confounding variables.

Separate linear regression modelling in smokers found no association between alcohol consumption and weight gain (regression coefficient: 0.005, 95% confidence intervals: -0.037, 0.046, (p=0.827). There was no evidence that this effect differed by gender, p for the interaction term was 0.728 or baseline BMI, p for the interaction term was 0.911. Cook's distance did not exceed 1 (min <0.001, max 0.03).

Variability of weight change in quitters according to baseline alcohol consumption

There was a significant, negative, linear relationship between weight change and alcohol consumption in quitters (p=0.015, r^2 0.070). For every additional unit of alcohol consumed per week at time of quitting, mean weight gain over 8 years was -0.174kg [-0.315, -0.034] (Figure 1). This was strengthened slightly after adjusting for confounding variables (regression coefficient= -0.180, p=0.011). Use of higher order terms did not improve the fit of this model. Cook's distance did not exceed 1 (min <0.001, max 0.087).

There was no evidence that this effect differed by gender, p for the interaction term was 0.909. The estimated regression coefficient was -0.164 in men and -0.229 in women.

Variability of weight change in quitters according to baseline alcohol consumption and BMI

We have previously demonstrated that 11% of the variability in weight gain in quitters was accounted for by a J-shaped curve with baseline BMI [Lycett et al, 2011]. There was no evidence that the association between alcohol and weight gain was modified by baseline BMI; p for interaction was 0.290. The effects of BMI and alcohol consumption are therefore independent, together they account for 17% of the variability of weight gain in quitters (Table 2). The regression lines for mean population weight gain according to BMI at different levels of alcohol consumption are plotted (Figure 2).

Discussion

Findings

Quitters who were successfully quit for 8 years gained 8.8kg. For every unit of alcohol they consumed at baseline they weighed 0.174kg less 8 years later than those who did not drink. This equates to people drinking alcohol at the maximum UK recommended weekly intake for women (14 units or 112g ethanol/week) would weigh a mean 2.4kg less than those who did not drink. This association was not significantly different in females and males. The effect was independent of baseline BMI.

Smokers weighed 2.2kg more than at baseline. We found no linear association between alcohol consumption at a quit attempt and weight gain in smokers. This was not modified by gender or baseline BMI.

The Role of Bias

We need to consider a number of sources of information bias in order to interpret the association we found between baseline alcohol consumption and weight gain in quitters. The strength of this study lies in the long-term follow up and the determination of continuous smoking abstinence, which was biochemically verified at each time point. This is in contrast to studies that rely on self report and point prevalence abstinence; these may overestimate quit rates and thus underestimate weight gain associated with quitting [Klesges et al, 1989, Klesges et al, 1997].

Although smoking status was well characterised, weight was self-reported in all at follow up and in a small group at baseline. We found no significant differences between measured and self reported weight at baseline or weight change over 8 years, we described these findings in a previous publication [Lycett et al, 2011]. However we cannot rule out the possibility that weight may have been underestimated, particularly in heavier individuals, for reasons of social desirability. Alcohol consumption was also self-reported and again may have been underreported for reasons of social acceptability. However underreporting of both measures would have not have changed the slope of our regression line. For underreporting to explain the association, those who underreported weight would have had to over-report alcohol consumption and this seems unlikely.

Alcohol was measured by careful questioning at baseline only. There is evidence that a single measure of alcohol consumption is a reasonable estimate of average alcohol consumption over several years. The Nurses' Health Study shows a high correlation between alcohol intake at a single point in time and alcohol intake over the following 6 years (R=0.75) [Giovannucci et al, 1991]. Also there is evidence from a large cohort that alcohol consumption does not change as a consequence of quitting smoking [Murray et al, 1996].

Only 52% of the participants enrolled in the original trial responded at eight year follow up. This is expected in smoking cessation trials where many people try to stop smoking but relapse and hence are embarrassed to respond to follow-up. Success in quitting was the factor most strongly associated with responding. If non response were to account for our results non-responders would have gained more weight the more alcohol they consumed at

baseline. While this remains a possibility as this was primarily a smoking cessation study there is no reason to believe that a participant's perception of weight gain or alcohol consumption influenced their decision to complete the questionnaire.

The Role of Confounding

It is possible that confounding explains the association. As this was a smoking cessation trial analyses on weight change were not planned; consequently behaviours such as diet and physical activity were not assessed. It is possible that those who drank more alcohol at baseline also had better dietary behaviour and did more physical activity than those who drank less. There is cross sectional evidence which shows a positive correlation between moderate alcohol intake and habitual physical activity ($r=0.41$ $p<0.01$)[Westerterp et al, 2004], but prospective studies have reported higher alcohol consumption is associated with lower weight gain after adjustment for physical activity and diet [Wannamethee & Shaper, 2003, Wannamethee et al, 2004, Wang et al, 2010]. To explore this in an ex-smoking population we carried out regression analysis on data from the Health Survey for England (HSE).

The HSE generates a cross sectional dataset from annual surveys on the health and lifestyle of a large number of people in England. For maximum compatibility with our own data set of abstainers we carried out our analysis on a subset of white ex-smokers. The distribution of socioeconomic class was similar between both sets of data. We used the 1998 HSE dataset as this was the nearest year to our cohort which contained comprehensive data on alcohol intake, physical activity and dietary patterns in over 4000 ex-smokers. We excluded those classified as problem drinkers.

We found in unadjusted analysis that an extra unit of alcohol drunk per week is associated with an average increase in 2.6 minutes of activity each week, including an extra 0.03 days a month doing 20 minutes or more, moderate to vigorous physical activity. So someone consuming 14 units of alcohol a week compared to no alcohol may be doing an extra 36 minutes of activity each week, including an extra 0.4 days a month doing 20mins of moderate or physical activity. If we consider a 68kg person walking briskly for 1 hour would burn approximately 320kcal, an extra 36 minutes of similar intensity exercise each week would burn an extra 192 kcal or theoretically an extra 27kcal each day. A daily deficit of 550kcal is required for a 0.5kg weight loss over 1 week. So an extra 27kcal expended daily could account for a theoretical weight change of 0.02kg each week. Based on this reasoning over 8 years one might expect an 8.3kg difference in weight to be seen, but this is not born out in epidemiological studies. Current available evidence suggests that even the recommendation of 30 minutes physical activity a day for cardiovascular fitness, is insufficient to prevent weight gain and 45 to 60 minutes moderate intensity activity each day is considered necessary[Saris et al, 2003, Wareham et al, 2005]. Given this, the extra 36 minutes of activity a week associated with moderate alcohol consumption is unlikely to account for the impact of alcohol on weight.

We analysed 9 indicators of a healthy diet from food frequency questions. There was a statistically significant association with all of these, although the sizes of these associations were too small to be meaningful and the directions of these associations were inconsistent. Some indicators showed alcohol consumption was associated with a less healthy diet,

namely more frequent salt use, red meat and fried food consumption, a greater amount of total fat and less frequent fruit and vegetable consumption. Other indicators found alcohol consumption was associated with a more healthy diet, namely more fibre and less frequent consumption of chocolate/crisps/biscuits and cakes. The largest association was seen for cake consumption, which was equivalent to one additional unit of alcohol resulting in an odds ratio of 1.013 (or a 0.013 [0.008, 0.017] unit decrease in the log of odds or of consuming cake less frequently (on a scale of 1 to 5).

Therefore although we did not measure physical activity and diet in our cohort, these findings suggest that they are unlikely to be confounding the association we found. However further studies which measure and adjust for these within the same study population are needed before we can be certain.

Our measure of alcohol consumption gave an estimate of total quantity of alcohol consumed on a weekly basis but no detail on the pattern of drinking. As mentioned in the introduction consideration of drinking pattern may have helped to explain our findings further.

Consistency of findings

In a cohort study of smokers and quitters, spanning 2 to 4 years, quitting smoking was associated with an increase in BMI, higher alcohol consumption, measured at one point in time, attenuated this rise in BMI. When investigated there was no evidence of effect modification by smoking status. However smoking status was self reported and quitting date was variable (any time after study entry), so a smaller weight gain may have diluted the

results. That study may also have lacked power to detect an effect modification because there were only 65 quitters and alcohol consumption was a binary variable [Froom et al, 1999].

A 2 year cohort found a small but significant inverse effect of alcohol consumption at the time of quitting and subsequent weight gain female smokers and quitters. This effect was seen after adjustment for physical activity[Kawachi et al 1996]. There was no investigation of interaction by smoking status. Abstinence was measured by point prevalence which may have underestimated weight gain and accounted for the small effect size seen.

Our findings are similar to the results from the Lung Health Study [Nides et al, 1994] which followed biochemically validated continuous abstainers for 5 years. The data showed that for each standard US drink per week consumed at baseline, the regression coefficient was -0.098 (p=0.02) in men and -0.234 (p=0.02) in women. (Although the Lung Health Study team reported these coefficients separately for each gender, there is no statistically significant difference between them according to our calculations). Equating these to UK units gave regression coefficients of -0.083 and -0.132 for men and women respectively. These values fit within the 95% confidence intervals our mixed sample but suggest a slightly lower effect than we found. The Lung Health Study also measured self reported alcohol consumption at baseline only and did not measure or adjust for physical activity or diet.

Thus there is consistent epidemiological evidence that alcohol consumption is associated with lower weight gain in people who stop smoking. We have been unable to find contrary evidence. We found no effect of gender despite epidemiological evidence in the general

population suggesting that one exists. However it may be that the effect in quitting smokers is stronger than in the population as a whole and as such the gender differences become negligible.

Biological Plausibility

We therefore have to consider the possibility that this is causal. Evidence for causality is bolstered by biological plausibility. Given the inconsistent effect of alcohol in epidemiological studies may be explained by variable induction of the MESO, it is possible that quitting smoking or the withdrawal of nicotine may induce this pathway.

Conclusion

A complex relationship exists between alcohol consumption and weight gain. In quitting smokers advice to reduce alcohol consumption may promote rather that prevent weight gain. However studies are needed which measure the amount and pattern of alcohol consumption reliably and prospectively in addition to an adequate measure and adjustment for potential confounders. These studies need to investigate the mechanisms of alcohol metabolism in quitting smokers and weigh the adverse health consequences of increasing alcohol against the benefit of smaller weight gain. Increasing alcohol should not currently be advised for preventing weight gain during smoking cessation.

Declaration of Interests

Paul Aveyard has done consultancy work on smoking cessation for Pfizer, McNeil, and Xenova Biotechnology. Marcus Munafò has received fees for invited lectures from the National Health Service, GlaxoSmithKline, Novartis, the Moffitt Cancer Research Center and

the Karolinska Instituet, and received benefits in kind (hospitality etc.) from various pharmaceutical companies. He has received research and travel support from the European Research Advisory Board, GlaxoSmithKline, Pfizer Consumer Healthcare and Novartis. Consultancy has been provided to the European Commission, The American Institutes for Research, the National Audit Office and G-Nostics Ltd. Elaine Johnstone has received consultancy income from European Network for Smoking Prevention. Michael Murphy has received consultancy income from the European Network for Smoking Prevention and has provided scientific consultancy services through the University of Oxford ISIS Innovation to the National Audit Office and G-Nostics Ltd.

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Table 1 Baseline characteristics by smoking status

Variable	smokers	quitters
n*	613	85
SES (%)		
I (professional)	2.5	1.2
II (managerial/technical)	31.1	33.3
III (skilled, non-manual)	21.9	16.7
III (skilled, manual)	21.0	28.6
IV (partially skilled)	17.4	15.5
V (unskilled)	6.2	4.8
HR score (mean(SD))	15 (5)	14 (4)
Cigs per day (mean(SD))	24 (7)	23 (7)
Weekly units alcohol (mean(SD))	10.1(13.03)	9.15(9.62)
max	86	43
min	0	0
IQR	11	13
median	6	7
Height (cm)	168 (9)	169 (9)
Age(mean(SD))	42 (10)	46 (11)
BMI (mean(SD))	25 (4)	24 (4)
Ethnicity (%):		
European	98.2	100
Indian/Pakistani/Bangladeshi	1.0	0
Other	0.7	0
Not stated	0.2	0
Active patch treatment (%)	48.6	57.6
Women (%)	59.4	58.8

*data was not available in all categories for every person, alcohol consumption reported in 596 smokers and 84 quitters.

Table 2. Regression Analysis of weight change according to baseline BMI and alcohol consumption in quitters

Variable	Regression coefficient*	Lower 95%CI	Upper 95%CI	p value	r^2
Constant	7.886	6.360	9.412	<0.001	0.172
BMI#	0.217	-0.175	0.609	0.274	
BMI^{2} #	0.062	0.006	0.118	0.030	
Alcohol units/week#	-0.161	-0.296	-0.026	0.020	

*Stepwise adjustment, using p=0.2 cut-off for confounding variables of baseline height, BMI, Horne Russell Score, daily number of cigarettes, weekly units of alcohol at baseline, age in years, socio-economic status and ethnic origin.
Re-centred around the total sample mean.