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Impact of Lipid-lowering and Anti-hypertensive Pharmacotherapy on Health-related Behaviors

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ABSTRACT: The widespread use of lipid-lowering drugs and anti-hypertensives has become central to the prevention of cardiovascular disease (CVD) and type 2 diabetes mellitus. However, their influence on individual health behaviors remains unclear. This review focuses on how preventive pharmacotherapy affects both positive health behaviors, including diet and physical activity, as well as negative health behaviors, including smoking and alcohol use. This review evaluates whether these medications function primarily as complements to or substitutes for lifestyle modification. Across diverse study designs and populations, the evidence suggests that preventive pharmacotherapy does not uniformly reinforce healthy behaviors. These findings carry significant implications for clinical practice and public health policy. As reliance on preventive pharmacotherapy continues to expand, medications should not be positioned as replacements for lifestyle change. Instead, as recommended by the World Health Organization, pharmacologic treatment should be integrated systemically with lifestyle changes to ensure that reductions in CVD and diabetes risk are sustained through both biomedical and behavioral pathways.

KEYWORDS: Social and Behavioral Science, Health Economics, Behavioral Economics, Pharmacotherapy.

Introduction

According to the World Health Organization, from 1990 to 2022, the number of individuals diagnosed with diabetes mellitus rose from 200 million to 830 million.[1] In 2021, diabetes was the direct cause of 1.6 million deaths, as well as the cause of 530,000 deaths by kidney disease.[1] From 1990 to 2023, the number of individuals diagnosed with cardiovascular disease (CVD) rose from 311 million with 13.1 million deaths to 626 million with 19.2 million deaths, making CVD the leading cause of death globally.[2] In 2021, high blood glucose from diabetes caused around 11% of cardiovascular deaths.[1]

Lifestyle and dietary modifications have always been essential to the primary and secondary prevention of diabetes mellitus and cardiovascular disease (CVD).[3-6] Smoking cessation, decreased consumption of alcohol, increased physical activity, and decreased intake of saturated fats are all associated with lower risks of CVD and type 2 diabetes mellitus.[7-10] In the past few decades, the use of lipid-lowering drugs (LLDs) and anti-hypertensives (AHs) as a complement to lifestyle modifications has become prevalent,[11-13] and will continue to expand due to the implementation of recent policies.[3,4,14]

The relationship between the initiation of LLDs and AHs remains unresolved.[15] Therefore, clinicians should precede the prescription of preventative pharmacotherapy with lifestyle modification.6 Following initiation, the perceived effectiveness of the drugs may incentivize patients to live a healthier lifestyle.[16,17] However, patients may see pharmacotherapy as a replacement rather than a complement to lifestyle modification,[17] and use statins as a "get out of jail free card" to engage in unhealthy behaviors;[18] thereby reducing the effectiveness of pharmacotherapy.[19,20] Furthermore, clinicians may not stress the importance of lifestyle modification after initiation.[21]

The objective of this review is to synthesize the empirical evidence on the effects of LLDs and AHs on health behaviors such as diet, exercise, smoking, and alcohol use. By integrating findings from repeated cross-sectional and longitudinal cohort studies, as well as fixed-effects and instrumental-variable designs, this review evaluates whether preventive pharmacotherapy functions primarily as a complement to or a substitute for lifestyle modification. It also explores how these effects can vary by sex, study design, and clinical context. The clarification of these behavioral responses will be essential for optimizing prevention strategies and aligning pharmacologic treatment with sustained lifestyle change in the prevention of CVD and type 2 diabetes mellitus.

Changes in Diet and Caloric/Fat Intake:

Dietary composition and total energy intake are both vital in the prevention of CVD and type 2 diabetes mellitus. Excess caloric consumption and high saturated fat intakes promote dyslipidemia, insulin resistance, and weight gain.[22-24] Consequently, dietary modification in the form of reductions in total calories and saturated fat is routinely emphasized as a first-line intervention for individuals with hyperlipidemia and elevated cardiovascular risk.[25-28] Statins and other LLDs are also highly effective at reducing LDL cholesterol;[29-31] although, unlike cholesterol, they do not directly address excess energy intake or diet quality, thereby raising the possibility that pharmacologic lipid control may alter incentives for sustained dietary restraint.[17]

In this section, we review two studies related to changes in diet, caloric intake, and fat intake. One study by Sugiyama *et al.* focuses on the effects of statin use on total cholesterol and different metrics of body composition (BMI, overweight,

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and obesity).[32] Another study by Kaestner *et al*. focuses on differences in caloric and fat intake between statin users and non-users.[17]

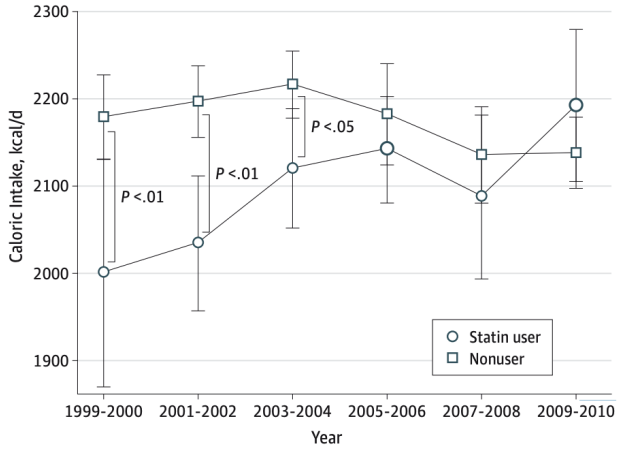

Figure 1: Relationship of caloric intake estimates between statin users and non-users. Statin users had significantly lower caloric intake than non-users from 1999-2004; however, in the later years (2005-2010), the difference between the two groups decreased significantly. Adjusted for age category, sex, race and ethnicity, educational attainment, and diabetes diagnosis. Error bars represent 95% CIs. Larger points represent significant changes from 1999-2000. Note. Reproduced from Sugiyama *et al*.[32]

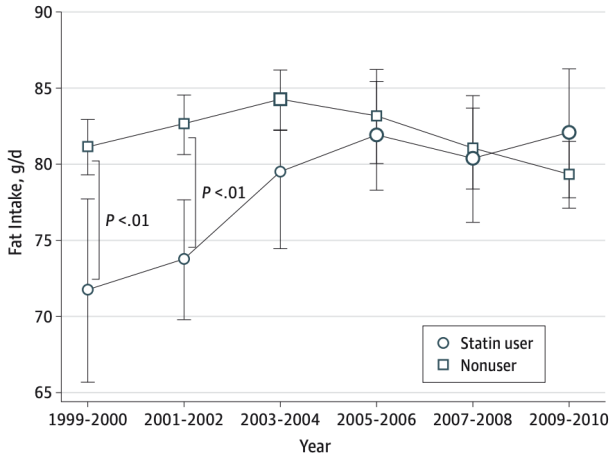

Figure 2: Relationship of fat intake estimates between statin users and non-users. Statin users had significantly lower fat intake than non-users from 1999-2004; however, in the later years (2005-2010), the difference between the two groups decreased significantly. Adjusted for age category, sex, race and ethnicity, educational attainment, and diabetes diagnosis. Error bars represent 95% CIs. Larger points represent significant changes from 1999-2000. Note. Reproduced from Sugiyama *et al*.[32]

According to the work by Sugiyama *et al*.,[32] statin users had significantly lower calorie (Figure 1) and fat (Figure 2) intake than non-users from 1999-2004; however, in the later years (2005-2010), the difference between the two groups decreased significantly. This change is explained by the steep increase in the use of Statins, as users almost doubled from 1999 to 2010. Additionally, a cross-sectional study by Lofgren *et al*. that was conducted in Rhode Island in the 2000s found a decrease in caloric intake in elderly Statin users that was insignificant.[33] Another cross-sectional study by Lytsy *et al*.,[34] which was conducted in Sweden in 2004, concluded that Statin users were more likely to avoid food with higher levels of fat content than non-users. On the other hand, a longitudinal study by Mann *et al*. in Veterans Affairs primary care clinics followed new statin users and found no increase in caloric and fat intake.[35] It is important to keep in mind that this study only took place for 6 months, and although longitudinal studies allow for more certainty in causal inferences, the cross-sectional study from Figures 1 and 2 utilized data from 12 years, allowing them to see time-trends when there was a steep increase in the use of Statins.

Sugiyama *et al*. explain the shift through two mechanisms.[32] One possibility is that following the initiation of Statin, especially after users see improvement in their LDL-C levels, users may lose the incentive to make positive health-related lifestyle modifications. Physicians may have also contributed to this by making the main focus of the appointment on the administration of statins rather than on lifestyle modification. Another possibility is that as the prescription of statins increased, those who didn't want to change their diet initiated statins, and those who didn't prefer to initiate statins instead made positive lifestyle modifications.

According to Kaestner *et al*.,[17] following the initiation of statin, total cholesterol decreased significantly. As shown in Table 1, for males, the decrease in total cholesterol following initiation of statin therapy was 44 points, and for females, it was 49 points. These results reflect the previously established efficacy of statin use for lowering cholesterol, specifically in a non-experimental context.[32] Also, based on their study, the initiation of statin is associated with a statistically significant increase in BMI of 0.4 units for males, and a 0.3 to 0.5 unit increase for females. Although these results were significant, they were still relatively small (about 10% of a standard deviation). However, the results for obesity suggested that statin initiation is associated with about a 20% increase in obesity for males and a 33% increase for females. Estimates related to being overweight are relatively small and statistically insignificant. Ultimately, estimates in Table 1 provided strong and consistent evidence of the association between statin initiation with a small increase in BMI and a relatively large increase in obesity, suggesting a worsened diet following initiation of statins.

Table 1: Estimates of the effect of statin use on total cholesterol and weight in the full sample, including the low-cholesterol group.*

	Total Cholesterol		BMI		Overweight		Obese	
	Males	Females	Males	Females	Males	Females	Males	Females
Fixed-effects Estimates	−46.38** (1.65)	−50.68** (2.17)	0.36** (0.11)	0.44** (0.17)	0.02 (0.02)	0.00 (0.02)	0.04** (0.02)	0.06** (0.02)
Fixed-effects Estimates w/ separate trends	−38.54** (2.36)	−44.25** (3.43)	0.41** (0.13)	0.32*** (0.18)	0.01 (0.02)	0.00 (0.02)	0.05*** (0.03)	0.05 (0.03)
P-value Test of Diff. Trends	0	0	0.72	0.82	0.14	0.36	0.8	0.65
Fixed-effects IV Estimates	−52.49** (1.85)	−55.04 (2.99)	0.33 (0.17)	0.52** (0.25)	0.02 (0.02)	0.00 (0.03)	0.04 (0.03)	0.08** (0.03)
P-value Over ID Test	0.02	0.01	0.18	0.52	0.91	0.53	0.51	0.27
Baseline Mean/Std. Dev.	36.13	44.59	3.47	4.95	0.81	0.61	0.27	0.18

Following the initiation of statin therapy, total cholesterol decreased significantly, while BMI and obesity increased significantly.* The sample size is approximately 6,500 (1,300 unique people) males and 7,000 (1,400 unique) females. Sample sizes differ slightly for each variable because of missing

values. Regression models include individual fixed effects, dummy variables indicating interview/exam, age, marital status, whether a person had a cardiovascular event (CVD), whether parents had CVD, and whether parents are living. ** indicates p-value ≤ 0.05, *** indicates 0.05 < p-value ≤ 0.10. Note. Adapted from Kaestner *et al.*[17]

The relationship between statin use and dietary behavior differs between the study designs by Sugiyama *et al.* and Kaestner *et al.*,[17,32] but converges on a common theoretical interpretation. Sugiyama *et al.* used repeated cross-sectional NHANES data,[32] which revealed that early dietary restraint among statin users decreased over time as statin use expanded, closing the gap in caloric and fat intake between users and non-users; this pattern was consistent with population-level risk compensation. However, Kaestner *et al.* used longitudinal fixed-effects data,[17] which, in turn, revealed that statin initiation caused large reductions in cholesterol while simultaneously producing small increases in BMI and substantial increases in obesity risk. This indicated that dietary behavior worsened within individuals after treatment began. Altogether, the cross-sectional time trends identified by Sugiyama *et al.* and the causal within-person effects estimated by Kaestner *et al.* both support the conclusion that statins often function as substitutes for dietary modification rather than complements. This shows how improvements in biomedical risk can reduce incentives for sustained lifestyle change.[17,32]

Changes in Physical Activity Levels:

Consistent physical activity is essential to cardiometabolic prevention.[36] It carries well-established benefits for lipid profiles, blood pressure, insulin sensitivity, and body weight regulation.[37-39] Increased physical activity for individuals with hyperlipidemia or hypertension is recommended by clinical guidelines,[28] both prior to and following the initiation of pharmacologic therapy. Relative to dietary change, the physical activity requirement for sustained behavioral effort and ongoing motivation makes it particularly vulnerable to perceived reductions in disease risk after initiation of medication.

In this section, we review three studies related to changes in physical activity levels in LLD users and individuals diagnosed with diabetes mellitus. The study by Oh *et al.* examined the difference in physical activity levels in MET between LLD users and non-users in individuals who had hyperlipidemia.[40] The work by Kaestner *et al.* estimated the effects of statin use on both sedentary and vigorous physical activity levels.[17] A third study by Schneider *et al.* examined the change in physical activity in MET following a diabetes diagnosis.[41]

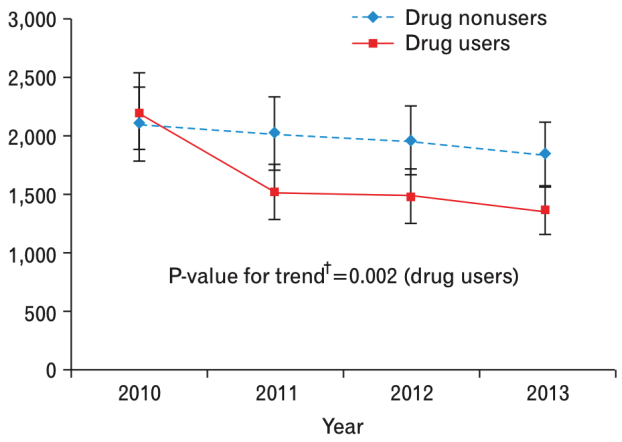

Figure 3: Difference in physical activity levels between LLD users and nonusers in individuals with hyperlipidemia. Drug users showed a statistically significant decrease in physical activity when compared to non-users in the years 2010-2013. Adjusted for sex, age category, level of education, marital status, body mass index, and diagnosis of diabetes. † P for trend was using a general linear model in complex sample analysis. Note. Reproduced from Oh *et al.*[40]

In the study by Oh *et al.*,[40] physical activity was assessed using the Korean version of the International Physical Activity Questionnaire (IPAQ)[42,43] The following points were assessed in IPAQ: the duration (minutes per day) and frequency (days per week) of vigorous, moderate-intensity, and walking activities. The number of hours was weighted by the specific metabolic equivalent (MET) score for each activity.

1) Vigorous MET (min/wk) = 8.0 × vigorous - intensity activity (min/d) × vigorous (d/wk)

2) Moderate MET (min/wk) = 4.0 × moderate - intensity activity (min/d) × moderate (d/wk)

3) Walking MET (min/wk) = 3.3 × walking (min/d) × walking (d/wk)

4) Total MET (min/wk) = sum of vigorous + moderate + walking MET scores)

According to Figure 3, when MET scores were adjusted for all variables, drug users showed a statistically significant decrease in physical activity when compared to non-users in the years 2010-2013. According to the author,[40] the therapy didn't include or emphasize the necessary lifestyle modifications that are needed in addition to medication. This may reduce the impact of standalone pharmacological therapy.

According to Kaestner *et al.* FEIC Estimates demonstrated a statistically significant decrease of 17% (of a standard deviation) in sedentary activity and a statistically significant increase of 24% in vigorous activity,[17] as shown in Table 2. Additionally, statin use is associated with a statistically significant increase of 18% (of a standard deviation in sedentary activity among females. The author concluded that evidence is mixed on whether statin use is complementary to changes in physical activity. They explained that it is entirely possible that other changes may have occurred simultaneously with statin use, such as greater contact with medical providers.

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Table 2: Estimates of the effect of statin use on sedentary and vigorous physical activity by sex.*

	Sedentary (M)	Sedentary (F)	Vigorous (M)	Vigorous (F)
Fixed-Effects Estimates	-0.47*** (0.26)	0.53*** (0.27)	0.40** (0.17)	-0.01 (0.16)
Fixed-Effects (Separate Time Trends)	NA	NA	NA	NA
P-value (Test of Diff. Trends)	NA	NA	NA	NA
Baseline Mean	3.05	2.76	1.71	1.4
FEIV Estimates	-0.48*** (0.27)	0.65** (0.28)	0.35*** (0.19)	-0.01 (0.16)
P-value (Over-ID Test)	NA	NA	NA	NA

FEIC Estimates demonstrated a statistically significant decrease of 17% (of a standard deviation) in sedentary activity and a statistically significant increase of 24% in vigorous activity. * The sample size is approximately 4,500 (939 unique people) males and 4,500 (926 unique) females. Sample sizes differ slightly for each variable because of missing values. Regression models include individual fixed effects, dummy variables indicating interview/exam, age, marital status, whether a person had a cardiovascular event (CVD), whether parents had CVD, and whether parents are living. ** indicates p-value≤0.05, *** indicates 0.05 < p-value ≤ 0.10. Note. Adapted from Kaestner *et al*.[17]

Table 3: Change in physical activity* for participants with reported diabetes compared to those without diabetes.

	Crude N=84,300			BMI adjusted N=83,324			Full model*** N=76,020		
	Beta	SE**	p-value	Beta	SE	p-value	Beta	SE	p-value
Walking MET hours/week	0.259	0.074	<0.001	0.23	0.075	0.002	0.263	0.078	<0.001
Mild MET hours/week	0.025	0.056	0.66	0.027	0.057	0.632	0.027	0.06	0.649
Moderate MET hours/week	0.091	0.09	0.312	0.071	0.091	0.433	0.095	0.096	0.321
Vigorous MET hours/week	0.239	0.118	0.043	0.189	0.119	0.113	0.2	0.125	0.11
Total MET hours/week	0.613	0.176	<0.001	0.517	0.178	0.004	0.585	0.186	0.002
Episodes ≥20 min/week	0.234	0.054	<0.001	0.217	0.054	<0.001	0.26	0.057	<0.001

A diabetes diagnosis is associated with statistically significant but modest increases in physical activity. * Computing change from earlier time point for those missing data (up to 1 year earlier, for instance if missing year 4 then change at year 5 is year 5 minus year 3) ** SE- standard error *** Full model included year, age, ethnicity, BMI, education (years), family history of diabetes, physical functioning (SF-36), pain (SF-36), energy/fatigue (SF-36), social functioning (cut at 50), depression (CES-D, cut at 0.06), number of chronic diseases and strenuous/hard exercise at age 18 years. Note. Adapted from Schneider *et al*.[41]

Table 3 from Schneider *et al*. examined changes in physical activity following a new diabetes diagnosis using longitudinal data from the Women's Health Initiative Observational Study: a study that followed 84,300 postmenopausal women from the age of 50 to 79 for up to seven years.[41] Through the use of linear mixed models with sequential adjustment for BMI and a comprehensive set of sociodemographic, health, and functional covariates, the study estimated within-person changes in activity among women with a diabetes diagnosis compared with those without. The results show that a diabetes diagnosis is associated with statistically significant but modest increases in physical activity; these changes were driven primarily through walking and activity frequency. In the fully adjusted model, women with a new diabetes diagnosis increased total physical activity by approximately 0.6 MET-hours per week ($\beta \approx 0.59$), walking by about 0.26 MET-hours per week, and the number of ≥20-minute activity episodes by roughly 0.26 episodes per week. However, changes in mild and moderate activity were not statistically significant. The initial association with vigorous activity (≈0.20 MET-hours per week) attenuated after full adjustment, and no significant change in sedentary time was observed. Therefore, Table 3 shows that, at least among postmenopausal women, a diabetes diagnosis prompted small but consistent increases in overall activity (equivalent to only about 6% of recommended weekly physical activity levels), rather than broad shifts toward higher-intensity exercise or reduced sitting.

The contrast in findings between Oh *et al*. and Kaestner *et al*. regarding physical activity among LLD users is largely caused by the differences in study design, population composition, and the clinical context in which the intervention was experienced.[17,40] Oh *et al*. used data from the Korean National Health and Nutrition Examination Survey (2010-2013), which utilized a repeated cross-sectional design.[40] This study found a statistically significant 38% decrease in total physical activity among LLD users, which was measured in MET-minutes per week, while it found no significant change among non-users. Due to the study's examination of population-level trends rather than within-person behavioral change and its lack of stratification of analyses by sex, the observed decline likely reflects a net effect driven by female participants, among whom statin use may function as a substitute for lifestyle modification.

In contrast, Kaestner *et al*. utilized a study design with longitudinal data with individual fixed-effects and instrumental-variable methods and identified sex specific responses.[17] Men reduced sedentary behavior and increased vigorous physical activity following statin initiation, while women increased sedentary behavior and exhibited no gains in vigorous exercise. However, these patterns should not be interpreted as evidence that women are inherently less responsive to health interventions. Evidence from other clinical contexts, such as studies of diabetes diagnosis among postmenopausal women, demonstrates that women are capable of increasing physical activity when the intervention provides a salient "teachable moment."[41] Rather, preventive pharmacological therapy for hyperlipidemia may attenuate perceived urgency and crowd out behavioral change, particularly among women, while functioning as a complement to lifestyle modification for men. Consequently, when these heterogeneous responses are aggregated in female-majority samples, such as that of Oh *et al*. They manifest as an overall decline in physical activity.[40] Taken together, these studies are not contradictory but illustrate that responses to statin therapy are context dependent: sex moderates behavioral change, but diagnosis severity and clinical framing ultimately determine whether treatment complements or substitutes for physical activity.

Changes in Smoking and Alcohol Use:

Smoking and the excessive consumption of alcohol are both major modifiable risk factors for CVD and Diabetes mellitus.[44-47] Smoking cessation is among the most effective behavioral interventions for reducing CVD risk.[48,49] Low to moderate consumption of alcohol has been associated with improved lipid profiles and insulin sensitivity,[50,51] and heavy consumption with increased cardiometabolic risk.[52] The strong relationship between smoking and alcohol use and socioeconomic status, health awareness, and healthcare engagement makes it so that studies examining these behaviors among users of preventative pharmacotherapy are particularly vulnerable to confounding by selection into treatment.[53]

In this section, we review three studies related to smoking and alcohol use. The study by Kinjo *et al*. examined changes in smoking, alcohol, and reported health in patients with hypertension or hyperlipidaemia who were recommended for pharmacological treatments, and whether they had initiated those treatments[54] The work by Kiortsis *et al*. examined LLD compliance in relation to smoking and alcohol use.[55] An additional study by Kaestner *et al*. examined the effects of statin use on smoking and alcohol use.[17]

Table 4: Smoking, alcohol, and unhealthy lifestyle changes are categorized by medication use in patients with hypertension or hyperlipidaemia who were recommended for pharmacological treatments.*

Characteristic	Hypertensive on AHs (N=8099)	Hypertensive w/o AHs (N=3752)	Hyperlipidaemic on meds (N=4645)	Hyperlipidaemic w/o meds (N=4550)
Non-current smoker (%)	6923 (85.5)	791 (21.1)	666 (14.3)**	866 (19.0)
Reported health excellent/very good (%)	2039 (25.2)**	1463 (39.0)	1231 (26.5)**	950 (20.9)
Alcohol <5 drinks (%)	7003 (86.5)**	848 (84.1)	693 (14.9)	3850 (84.6)

Relative to non-users, anti-hypertensive and lipid-lowering drug users were more often former/never smokers, and even after adjustment for age, gender, race, and comorbid conditions, AH and LLD users were more likely to be non-current smokers. * Adjusted for age, gender, race, diabetes, cardiovascular disease (angina, congestive heart failure, and cerebrovascular disease), and other comorbid conditions (cancer, arthritis, and chronic obstructive pulmonary disease). ** 8,099 antihypertensive users versus 3,752 non-users. Note. Adapted from Kinjo *et al*.[54]

As seen in Table 4, Kinjo *et al*. used data concerning smoking, alcohol, and reported health, which were assessed through cross-sectional surveys in the National Center for Health Statistics of the Centers for Disease Control in NHANES 1999–2000, 2001–2002, 2003–2004, 2005–2006, 2007–2008, and 2009–2010.[54] Relative to non-users, anti-hypertensive and lipid-lowering drug users were more often former/never smokers, and even after adjustment for age, gender, race, and comorbid conditions, AH and LLD users were more likely to be non-current smokers. AH users were more likely to be involved in healthy behaviors like the aforementioned lower use of tobacco, as well as lower use of alcohol; however, the association did decrease in the adjusted model.

Table 5: Smoking and alcohol use characteristics according to lipid-lowering drug compliance.

Variable	Whole study population (n=193)	High Compliance (n=81)	Intermediate Compliance (n=75)	Low Compliance (n=37)	P-value
Cigarettes per day	2.5 ± 6.8	1.7 ± 5.2	2.1 ± 5.8	5.0 ± 10.35*	0.0447
Pack-years	11.4 ± 16.4	12.0 ± 18.1	9.8 ± 14.7	13.7 ± 16.2	0.46
Alcohol (g/day)	7.4 ± 17	6.1 ± 16.4	8.9 ± 18.4	7.5 ± 14.8	0.58

Individuals who smoked more cigarettes per day and had a higher cumulative consumption (pack-years) showed lower compliance, relative to those who smoked less. * The high compliance group is patients who reported that they missed 0% of the prescribed pills, the intermediate group (missed less than 6% of the prescribed pills), and the low compliance group (missed 6% or more of the prescribed pills). Means and standard deviations are given. Note. Adapted from Kiortsis *et al*.[55]

This idea from Kinjo *et al*. is consistent with other research,54 including analyses by Kiortsis *et al*.,[55] which showed associations between AH and LLD non-adherence, immoderate alcohol use, and tobacco use.[56] Table 5 showed that individuals who smoked more cigarettes per day and had a higher cumulative consumption (pack-years) showed lower compliance, relative to those who smoked less.[55]

According to Kaestner *et al*.,[17] estimates for smokers and heavy smokers are mostly small and statistically insignificant for males, as shown in Table 6. Furthermore, estimates for females are less consistent in both sign and statistical significance. In terms of alcohol use, FE and FEIV estimates showed a 13% (of mean) and 21% increase in the probability of males being a moderate (>3 oz.) drinker; However, they showed effectively no association between initiation of statins and alcohol use for females.

Table 6: Estimates of the effect of statin use on smoking and drinking in the full sample, including the low-cholesterol group.*

Outcome	Smoker (M)	Smoker (F)	Heavy Smoker (M)	Heavy Smoker (F)	Drinker (M)	Drinker (F)	Alcohol >3 oz (M)	Alcohol >3 oz (F)
Fixed-Effects Estimates	-0.01 (0.02)	-0.02 (0.02)	-0.01 (0.01)	0.02* (0.01)	0.01 (0.02)	0.02 (0.02)	0.05** (0.02)	0.02 (0.02)
Fixed-Effects (Separate Time Trends)	-0.01 (0.02)	0.04*** (0.02)	-0.03*** (0.02)	0.04*** (0.02)	0.06** (0.03)	0.02 (0.04)	0.02 (0.03)	0.00 (0.02)
P-value (Test of Diff. Trends)	0.88	0.12	0.75	0.68	0.16	0.61	0.52	0.79
Baseline Mean	0.27	0.31	0.14	0.09	0.78	0.59	0.39	0.17
FEIV Estimates	-0.01 (0.03)	-0.06** (0.03)	0.01 (0.02)	0.00 (0.02)	-0.03 (0.03)	0.02 (0.03)	0.08** (0.03)	0.03 (0.02)
P-value (Over-ID Test)	0.71	0.84	0.7	0.54	0.69	0.32	0.76	0.9

Initiation of statin is generally not associated with statistically significant changes in smoking or general drinking; However, Alcohol consumption >3oz increases by a significant amount in men. Estimates for smokers and heavy smokers are mostly small and statistically insignificant for males. *The sample size is approximately 6500 (1300 unique people) males and 7000 (1400 unique) females. Sample sizes differ slightly for each variable because of missing values. Regression models include individual fixed effects, dummy variables indicating interview/exam, age, marital status, whether a person had a cardiovascular event (CVD), whether parents had CVD, and whether parents are living. ** indicates p-value≤0.05, *** indicates 0.05 < p-value ≤ 0.10 Note. Adapted from Kaestner *et al*.[17]

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Kinjo *et al*. and Kaestner *et al*. presented contrasting but complementary perspectives on smoking and alcohol behaviors among AH and LLD users.[17,54] This difference can largely be attributed to the differences in study design and analytic approach. Kinjo *et al*. analyzed cross-sectional NHANES data and found that AH and LLD users were more likely than non-users to be former or never smokers and moderate alcohol users.[54] This pattern held even after adjustment for demographic and clinical confounders and suggests a strong healthy user effect. The healthy user effect is where individuals who initiate preventive pharmacotherapy already engage in healthier behaviors or possess favorable socioeconomic characteristics that correlate with lower tobacco and alcohol use. Kaestner *et al*. instead analyzed longitudinal fixed-effects data and isolated within-person changes following statin initiation.[17] They found little evidence that statins causally reduced smoking and that smoking estimates are small and largely statistically insignificant for both sexes. Also, alcohol use increased modestly among men (13-21% increase in the probability of moderate drinking), but showed no meaningful association for women. These findings indicated that the healthier smoking and alcohol profiles observed among AH and LLD users in cross-sectional data primarily reflect selection into treatment rather than behavioral change induced by medication. Kinjo *et al*. captured baseline differences between users and non-users consistent with healthy user bias, while Kaestner *et al*. demonstrated that initiating statins does not substantially improve smoking behavior and may even coincide with risk compensation in alcohol consumption among men.[17,54] This reinforces the importance of distinguishing selection effects from causal behavioral responses in observational studies of preventive pharmacotherapy.

Kiortsis *et al*. shifted the question from "users vs non-users" to "how well do users adhere".[55] Within 193 treated hyperlipidemic outpatients, lower compliance clustered with higher cigarettes/day (low-compliance mean 5.0 vs high-compliance 1.7, $p \approx 0.045$), and they reported no meaningful sex difference in non-compliance overall. So, tobacco use appeared linked to poorer implementation of pharmacologic prevention, not just to whether someone is prescribed it.

Conclusion

This review synthesizes evidence from repeated cross-sectional surveys, longitudinal cohort studies, and quasi-experimental designs to evaluate how LLDs and AHs influence key health behaviors related to CVD and type 2 diabetes mellitus prevention. The findings consistently demonstrate that preventive pharmacotherapy does not uniformly reinforce healthy behavior. In several domains, it may function as a partial substitute for lifestyle modification rather than a complement.

These results have important implications for clinical practice and public health policy. As the use of preventive pharmacotherapy continues to expand globally, medications should not be positioned as substitutes for lifestyle modification, whether done so implicitly or explicitly. Pharmacologic treatment should instead be integrated with behavioral counseling that emphasizes diet, physical activity, smoking cessation, and decreased alcohol use as ongoing components of risk reduction. With such integration, improvements in biomedical markers may paradoxically coexist with stagnation or deterioration in underlying health behaviors. This would thereby limit the effectiveness of prevention strategies for CVD and type 2 diabetes mellitus.

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