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THE ROLE OF HMG-COA REDUCTASE INHIBITORS IN STABLE ANGINA ACROSS OCCUPATIONAL GROUPS: BASELINE CLINICAL, FUNCTIONAL, AND METABOLIC CHARACTERIZATION

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ABSTRACT

Background: Stable angina reflects chronic coronary atherosclerotic disease in which ischemic symptoms interact with hemodynamic, lipid, inflammatory, oxidative, and occupational risk factors. Objective: To characterize the pretreatment clinical, functional, and metabolic profile of patients with stable angina assigned to different therapeutic groups before evaluating HMG-CoA reductase inhibitor-based regimens. Methods: The baseline analysis included 150 participants: 30 controls and 120 patients with stable angina distributed equally across four clinical groups. Occupational categories included medical professionals, programmers, marketing specialists, and drivers. Assessment comprised angina frequency, nitroglycerin use, Holter electrocardiography, ambulatory blood pressure monitoring, bicycle ergometry, lipid profile, apolipoprotein B, C-reactive protein, malondialdehyde, superoxide dismutase, catalase, ceruloplasmin, and nitric oxide metabolites. Results: Compared with controls, patients had higher LDL-C, apoB, CRP, MDA, blood pressure, heart rate, arrhythmic burden, and daily ST-segment depression, together with lower HDL-C, antioxidant enzyme activity, NOx, exercise threshold, and exercise duration. LDL-C correlated positively with MDA (r = .41) and ST-depression depth (r = .34) and inversely with NOx (r = -.47). Occupational risk patterns differed, particularly for smoking, night shifts, and stress. Conclusion: Stable angina was associated with a coordinated atherogenic, inflammatory, oxidative, endothelial, and functional phenotype before treatment. These findings provide a balanced baseline for subsequent evaluation of atorvastatin-containing regimens but do not independently establish therapeutic efficacy.

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Keywords: *stable angina, HMG-CoA reductase inhibitors, atorvastatin, occupational risk, oxidative stress, endothelial dysfunction.*

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Introduction

Stable angina is a manifestation of chronic coronary disease in which myocardial oxygen demand intermittently exceeds coronary supply, commonly during physical or emotional stress. Contemporary guidelines emphasize that management should extend beyond symptom relief to comprehensive reduction of atherosclerotic risk, including intensive control of low-density lipoprotein cholesterol (LDL-C), blood pressure, smoking, and physical inactivity (Vrints et al., 2024; Virani et al., 2023). HMG-CoA reductase inhibitors (statins) are central to this strategy because lowering LDL-C reduces major vascular events

across a broad range of cardiovascular risk profiles (Cholesterol Treatment Trialists' [CTT] Collaboration, 2010, 2012). The causal relationship between cumulative LDL exposure and atherosclerotic cardiovascular disease further supports lipid modification in established coronary disease (Ference et al., 2017).

The burden of stable angina is nevertheless heterogeneous. Patients with similar attack frequency may differ substantially in blood pressure, ambulatory ischemia, exercise tolerance, apolipoprotein B (apoB), inflammatory activity, oxidative stress, and endothelial nitric oxide availability. ApoB reflects the number of circulating atherogenic particles and may provide information not fully captured by LDL-C alone (Soffer et al., 2024). Inflammation, often indexed by C-reactive protein (CRP), and oxidative processes may further contribute to endothelial dysfunction and atherosclerotic activity (Fu et al., 2020; Ridker, 2004; Simantiris et al., 2023). Studies of statin therapy in stable coronary disease have also examined ambulatory ischemia, exercise-induced ischemia, endothelial function, and inflammatory markers, suggesting relevance beyond a single lipid measurement (Bogaty et al., 2003; Deanfield et al., 2010; Stone et al., 2005).

Occupational exposures may add another layer of cardiovascular burden. Shift work is associated with increased cardiovascular risk, while sustained job strain has been linked to coronary heart disease (Torquati et al., 2018; Xu et al., 2015). These exposures are relevant when comparing medical professionals, programmers, marketing specialists, and drivers, whose work patterns differ in night duty, stress, sedentary behavior, and smoking. Accordingly, the present paper characterizes the pretreatment clinical, functional, lipid-inflammatory, oxidative, and endothelial profile of patients with stable angina across occupational categories. The aim is to establish baseline comparability and pathophysiologic context before evaluation of atorvastatin-containing regimens; no post-treatment efficacy conclusions are drawn from this baseline dataset.

Methods

Study Design and Participants

This paper reports the baseline phase of a comparative clinical study. The analyzed sample comprised 150 participants: 30 individuals in the control group (Group I) and 120 patients with functional class II stable angina in Groups II-V, with 30 patients in each clinical group. The stable-angina groups were similar in age and duration of ischemic heart disease before therapy. The source material identifies the planned clinical arms as conventional therapy (Group II), atorvastatin (Group III), coenzyme Q10 (Group IV), and atorvastatin plus coenzyme Q10 (Group V). All values analyzed in this manuscript were obtained before treatment; therefore, the study findings reported here characterize baseline status rather than therapeutic response.

The occupational distribution of the 120 patients with stable angina consisted of 33 medical professionals, 28 programmers, 25 marketing specialists, and 34 drivers. Occupational risk factors included smoking, hypertension, dyslipidemia, overweight, night-shift work, stress burden, and physical inactivity. The control group served as a physiological reference for clinical, functional, lipid, inflammatory, oxidative, and nitric-oxide-related measurements.

Clinical and Functional Assessment

Clinical assessment included the frequency of anginal attacks per week and weekly nitroglycerin requirement. Functional evaluation included daytime, nighttime, and maximum heart rate obtained by Holter electrocardiographic monitoring; the number and depth of ST-segment depression episodes; extrasystoles per hour; systolic and diastolic blood pressure on ambulatory blood pressure monitoring (ABPM); and graded exercise testing by bicycle ergometry. Bicycle-ergometry endpoints were threshold power in watts and exercise duration in minutes. Together, these measures were used to characterize symptomatic burden, hemodynamic load, daily ischemia, arrhythmic activity, and exercise tolerance.

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Laboratory Assessment

The lipid-inflammatory panel included total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), LDL-C, the atherogenic coefficient, apoB, and CRP. Oxidative and antioxidant status was evaluated using malondialdehyde (MDA), superoxide dismutase (SOD), catalase, and ceruloplasmin. Total stable nitric oxide metabolites (NOx) were used as an index of NO-dependent vascular regulation. This combined laboratory panel permitted an integrated assessment of atherogenic particle burden, low-grade inflammation, lipid peroxidation, antioxidant defense, and endothelial-related nitric oxide availability.

Statistical Analysis

Continuous variables were reported as mean ± standard deviation (M ± SD), and categorical variables as n (%). Analysis of variance was used for comparisons among Groups II-V where reported. Pearson correlation coefficients were calculated in the stable-angina cohort (N = 120), with 95% confidence intervals and exact p values. Control-group comparisons were interpreted using the significance levels stated in the source dataset. Because this paper is based on the supplied baseline report, no additional modeling, imputation, or post-treatment analysis was introduced.

Results

Baseline Demographic and Occupational Characteristics

The mean age was closely matched across the four stable-angina groups, ranging from 42.2 ± 4.2 to 43.4 ± 4.3 years. Duration of ischemic heart disease ranged from 2.9 ± 1.0 to 3.3 ± 1.0 years, and BMI ranged from 26.9 ± 2.0 to 27.6 ± 1.8 kg/m². No statistically significant differences were reported among the clinical groups for age, ischemic heart disease duration, or BMI. The control-group BMI was lower at 24.8 ± 1.7 kg/m², whereas the patient groups clustered near the overweight range.

Risk-factor distribution varied more clearly by occupation. Drivers had the highest smoking prevalence (61.8%), while medical professionals had the highest reported frequency of night shifts (81.8%) and stress burden (84.8%). Programmers also had high night-shift exposure (71.4%) and stress (75.0%). Marketing specialists showed lower overweight prevalence (36.0%) than the other categories but still had substantial stress (76.0%) and physical inactivity (40.0%). These patterns demonstrate that the stable-angina cohort was clinically comparable across treatment groups but not occupationally uniform in the exposures that could sustain cardiovascular risk.

Table 1

Occupational Risk-Factor Profile of Patients With Stable Angina

Occupation	N	Smoking	HTN	Dyslipidemia	Overweight	Night shifts	Stress	Inactivity
Medical professional	33	9 (27.3%)	19 (57.6%)	22 (66.7%)	19 (57.6%)	27 (81.8%)	28 (84.8%)	7 (21.2%)
Programmer	28	9 (32.1%)	13 (46.4%)	18 (64.3%)	16 (57.1%)	20 (71.4%)	21 (75.0%)	7 (25.0%)
Marketing specialist	25	8 (32.0%)	11 (44.0%)	15 (60.0%)	9 (36.0%)	17 (68.0%)	19 (76.0%)	10 (40.0%)
Driver	34	21 (61.8%)	19 (55.9%)	24 (70.6%)	16 (47.1%)	13 (38.2%)	23 (67.6%)	12 (35.3%)

Note. Values are n (%). HTN = hypertension. Occupational categories total N = 120.

Clinical and Functional Profile

Anginal attack frequency was similar across Groups II-V, ranging from 5.3 ± 1.4 to 6.0 ± 1.8 episodes per week (p = .348). Nitroglycerin requirement differed more noticeably, from 4.5 ± 1.1 to 5.5 ± 1.3 tablets per week (p = .018), indicating that medication use was not determined solely by attack frequency. Mean daytime heart rate was approximately 80-82 beats/min in the patient groups compared with 68.0 ± 5.9 beats/min in controls. Mean systolic blood pressure was approximately 149-152 mmHg in Groups II-V compared with 125.7 ± 8.0 mmHg in controls, and mean diastolic pressure was approximately 89-91 mmHg compared with 78.4 ± 4.2 mmHg in controls.

No clinically significant ST-segment depression was recorded in controls, whereas Groups II-V experienced 1.63-1.88 episodes per day with a mean depth of 1.37-1.57 mm. Extrasystoles were also more

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frequent in patients, ranging from 4.7 ± 2.2 to 6.4 ± 2.2 per hour versus 1.0 ± 1.1 per hour in controls; the between-group difference among the clinical groups was significant (p = .026 in the narrative report). Exercise tolerance was consistently reduced. Threshold power was 120.4 ± 12.7 W in controls but only 77.1-79.9 W in Groups II-V, while exercise duration decreased from 9.8 ± 1.1 minutes to approximately 6.3-6.4 minutes. The close similarity of Groups II-V on these functional measures supports baseline comparability before the planned therapeutic comparisons.

Table 2

Principal Baseline Deviations Relative to the Control Group

Parameter	Control	Groups II-V	Direction	p vs. control
LDL-C, mmol/L	2.40 ± 0.36	4.11-4.29	Higher	< .001
ApoB, g/L	0.75 ± 0.09	1.21-1.24	Higher	< .001
CRP, mg/L	0.91 ± 0.38	3.41-3.52	Higher	< .001
MDA, nmol/mL	16.6 ± 2.0	30.1-32.5	Higher	< .001
SOD, arb. units/mg Hb	66.8 ± 6.5	36.0-37.2	Lower	< .001
NOx, µmol/L	70.0 ± 5.4	39.0-41.7	Lower	< .001
ST-depression episodes/day	Not recorded	1.63-1.88	Higher	< .001
Bicycle threshold power, W	120.4 ± 12.7	77.1-79.9	Lower	< .001
SBP by ABPM, mmHg	125.7 ± 8.0	149.4-151.6	Higher	< .001

Note. Ranges summarize mean values across Groups II-V. ApoB = apolipoprotein B; CRP = C-reactive protein; MDA = malondialdehyde; NOx = stable nitric oxide metabolites; SOD = superoxide dismutase; SBP = systolic blood pressure; ABPM = ambulatory blood pressure monitoring.

Lipid and Inflammatory Profile

All stable-angina groups showed a pronounced atherogenic profile. Total cholesterol ranged from 6.21 ± 0.44 to 6.44 ± 0.42 mmol/L, compared with 4.38 ± 0.40 mmol/L in controls. LDL-C ranged from 4.11 ± 0.46 to 4.29 ± 0.45 mmol/L in the patient groups versus 2.40 ± 0.36 mmol/L in controls. HDL-C was reduced to approximately 0.91-0.93 mmol/L compared with 1.41 ± 0.16 mmol/L in controls. The atherogenic coefficient was 5.76-6.26 arbitrary units in Groups II-V, compared with 2.14 ± 0.38 in the control group.

ApoB values were tightly clustered at 1.21-1.24 g/L among the patient groups compared with 0.75 ± 0.09 g/L in controls. CRP showed an even larger relative departure, reaching 3.41-3.52 mg/L compared with 0.91 ± 0.38 mg/L. For LDL-C, HDL-C, atherogenic coefficient, apoB, and CRP, each clinical group differed significantly from controls at p < .001, while differences among Groups II-V were not significant. Thus, the therapeutic groups entered the study with a broadly comparable atherogenic-inflammatory burden.

Oxidative, Antioxidant, and NO-Related Profile

MDA was almost twice the control level, increasing from 16.6 ± 2.0 nmol/mL in controls to 30.1-32.5 nmol/mL in Groups II-V. In contrast, antioxidant defense markers were lower in patients. SOD decreased from 66.8 ± 6.5 to 36.0-37.2 arbitrary units/mg Hb; catalase decreased from 234.6 ± 11.9 to 156.2-161.6 mkat/L; and ceruloplasmin decreased from 257.9 ± 16.7 to 157.6-163.1 mg/L. NOx also fell substantially, from 70.0 ± 5.4 µmol/L in controls to 39.0-41.7 µmol/L in Groups II-V. Each of these patient-control differences was reported as significant at p < .001, whereas differences among the stable-angina groups were generally not significant.

The coordinated increase in lipid peroxidation, decrease in antioxidant activity, and reduction in NOx indicates that the baseline metabolic disturbance was multidimensional. Importantly, the descriptive parallelism between oxidative markers and functional ischemia should not be interpreted as causal without longitudinal or mechanistic evidence.

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Correlations Among Lipid, Oxidative, Endothelial, and Ischemic Measures

Correlation analysis in the 120 patients with stable angina identified several statistically significant associations. LDL-C correlated positively with MDA (r = .41, 95% CI [.25, .55], p < .001) and inversely with NOx (r = -.47, 95% CI [-.60, -.32], p < .001). MDA was inversely related to NOx (r = -.41, 95% CI [-.55, -.25], p < .001). LDL-C also correlated with ST-segment depression depth (r = .34, 95% CI [.17, .49], p < .001). The association between apoB and LDL-C was weaker (r = .22, p = .014), and CRP correlated weakly but significantly with MDA (r = .28, p = .002). These findings support statistical coordination among atherogenic burden, oxidative stress, endothelial-related NO metabolism, and functional ischemia while remaining insufficient to demonstrate causal direction.

Table 3

Pearson Correlations in Patients With Stable Angina

Variable pair	N	r	95% CI	p
LDL-C - MDA	120	.41	[.25, .55]	< .001
LDL-C - NOx	120	-.47	[-.60, -.32]	< .001
MDA - NOx	120	-.41	[-.55, -.25]	< .001
LDL-C - ST-depression depth	120	.34	[.17, .49]	< .001
ApoB - LDL-C	120	.22	[.05, .39]	.014
CRP - MDA	120	.28	[.11, .44]	.002

Note. N = 120 for all correlations. Correlations describe association and do not establish causation.

Discussion

The principal finding of this baseline analysis is that stable angina in the studied cohort was characterized by an integrated clinical and metabolic phenotype rather than by isolated episodic chest pain. The patient groups had a comparable frequency of anginal attacks, similar exercise impairment, and broadly similar degrees of ambulatory ischemia. At the same time, they showed substantially higher blood pressure and heart rate, a markedly atherogenic lipid profile, higher apoB and CRP, increased lipid peroxidation, reduced antioxidant activity, and lower NOx. This convergence is clinically relevant because chronic coronary disease is driven by cumulative atherosclerotic burden and modifiable risk factors, not only by the immediate frequency of symptoms (Vrints et al., 2024; Virani et al., 2023).

The lipid findings provide a strong rationale for evaluating HMG-CoA reductase inhibitor therapy in this population. LDL-C values of approximately 4.1-4.3 mmol/L and apoB values of approximately 1.2 g/L indicate a high burden of atherogenic cholesterol and particle number. Large-scale randomized evidence has established that the magnitude of cardiovascular risk reduction tracks the achieved reduction in LDL-C, including with more intensive statin therapy (CTT Collaboration, 2010, 2012). The broader causal evidence for LDL in atherosclerotic cardiovascular disease also supports interpreting these baseline concentrations as clinically important treatment targets rather than passive biomarkers (Ference et al., 2017). The relatively weak correlation between apoB and LDL-C in this cohort further illustrates that cholesterol mass and atherogenic particle number are related but not interchangeable constructs, consistent with contemporary expert guidance on apoB (Soffer et al., 2024).

The inflammatory and oxidative data extend this interpretation. CRP was approximately four times the control value, while MDA was nearly doubled and antioxidant markers were substantially reduced. CRP is widely used as an indicator of inflammatory activity in cardiovascular research, although it should not be treated as a direct causal mechanism solely on the basis of association (Fu et al., 2020; Ridker, 2004). Likewise, MDA is a commonly studied marker of lipid peroxidation, but oxidative biomarkers have methodological limitations and require cautious interpretation (Simantiris et al., 2023). In the present dataset, the positive LDL-C-MDA correlation and inverse LDL-C-NOx and MDA-NOx correlations are therefore best understood as evidence of a coordinated risk phenotype rather than proof that any single marker directly causes another.

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The functional findings are also consistent with prior investigations that examined statins in stable coronary disease using outcomes beyond serum cholesterol. Earlier trials and clinical studies evaluated exercise-induced ischemia, ambulatory ischemic burden, endothelial function, and inflammatory biomarkers during atorvastatin therapy (Bogaty et al., 2003; Deanfield et al., 2010; Stone et al., 2005). The present report does not reproduce those treatment findings because it contains baseline data only. Instead, it establishes that Groups II-V entered treatment with similar objective limitations: approximately 1.6-1.9 ST-depression episodes per day, threshold exercise power around 77-80 W, and exercise duration around 6.3-6.4 minutes. Such similarity reduces the likelihood that subsequent between-group differences would simply reflect large baseline imbalances in ischemic severity.

Occupational exposures deserve separate consideration. Medical professionals had the greatest night-shift and stress burden, programmers also showed frequent night work and high stress, and drivers had the highest smoking prevalence. Meta-analytic evidence indicates that shift work is associated with higher cardiovascular risk and that high job strain is associated with coronary heart disease (Torquati et al., 2018; Xu et al., 2015). These findings do not prove that occupational exposures caused the biochemical or functional abnormalities observed here, but they strengthen the rationale for incorporating work schedule, stress management, smoking cessation, and physical activity into comprehensive secondary prevention rather than relying on pharmacotherapy alone.

Implications for Atorvastatin and Coenzyme Q10 Treatment Comparisons

The source study was structured to compare conventional therapy, atorvastatin, coenzyme Q10, and atorvastatin plus coenzyme Q10 after this baseline assessment. The present analysis supports the clinical relevance of the atorvastatin-containing arms because LDL-C, apoB, inflammation, oxidative stress, and endothelial-related NO metabolism were all abnormal before therapy. However, baseline differences cannot establish whether atorvastatin improves angina frequency, ST-segment ischemia, exercise tolerance, MDA, NOx, or other outcomes in this specific cohort. Those questions require the post-treatment measurements and prespecified between-group analyses. Coenzyme Q10 has been investigated as an adjunct for statin-associated muscle symptoms, with meta-analytic evidence suggesting possible benefit in some trials (Qu et al., 2018), but the current baseline dataset provides no evidence regarding its efficacy for stable angina or oxidative-endothelial outcomes.

Limitations

Several limitations should be considered. First, this manuscript is restricted to the baseline phase of the study and therefore cannot estimate the therapeutic effect of HMG-CoA reductase inhibitors, coenzyme Q10, or their combination. Second, the source document does not provide recruitment dates, sex distribution, detailed inclusion and exclusion criteria, randomization procedures, medication washout information, or an ethics committee identifier; these items should be supplied before journal submission. Third, the occupational analysis is descriptive, and the study does not report multivariable adjustment for smoking, hypertension, shift work, stress, or physical inactivity. Fourth, correlations among LDL-C, MDA, NOx, CRP, and ischemic measures are cross-sectional and cannot establish temporal or causal relationships. Finally, several biomarkers, particularly MDA and antioxidant enzyme measures, are useful in research but are not routinely standardized clinical endpoints. These limitations do not negate the baseline differences, but they define the appropriate scope of interpretation.

Conclusion

Before therapy, stable angina was characterized by symptomatic and electrocardiographic ischemia, higher hemodynamic burden, reduced exercise tolerance, atherogenic dyslipidemia, elevated apoB and CRP, increased lipid peroxidation, weakened antioxidant defense, and reduced NOx. Groups II-V were broadly comparable at baseline, supporting later treatment comparisons, while occupational subgroups differed in smoking, night work, stress, and physical inactivity. Correlations among LDL-C, MDA, NOx, and ST-segment depression suggest an interconnected atherogenic-oxidative-endothelial

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phenotype without establishing causality. Post-treatment analysis is required to determine the efficacy of HMG-CoA reductase inhibition.

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