Effect of Atorvastatin on LDL & hs-CRP in a Selected Thai Population

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Background: LDL and hs-CRP are risk factors for vascular events and can be modified by Statin.
Objective: To evaluate the baseline hs-CRP of a certain Thai population who would need Atorvastatin, to evaluate the dose response of Atorvastatin toward LDL and hs-CRP level, and to evaluate the efficacy and safety of different types of Atorvastatin.
Material and Method: Subjects, who needed Statin therapy, were randomized to receive either 20 mg of Berlin®-Atorvastatin® or Pfizer®-Atorvastatin®. The cross over took place after 8 weeks of therapy and continued for 16 weeks. Baseline blood tests were compared to 8 and 16 weeks. The effect of two brands of 20 mg Atorvastatin toward serum lipid, LFT, muscle enzyme and hs-CRP were compared.
Results: One hundred and ten subjects aged between 20-75 years enrolled in the present study. Fifty-four and 56 patients were randomized to group A and B. Baseline total cholesterol, LDL, HDL, and TG were 251, 174, 55, and 160 mg/dl respectively. There was a wide variation of baseline hs-CRP level. One hundred and seven patients completed this 16 weeks study. Atorvastatin 20 mg lowered TC by 32%, LDL 44% and hs-CRP 10% at 16 weeks for the entire study (p < 0.003). The effect of either Atorvastatin the lipid profiles and hs-CRP were different. There was no significant change in LFT or muscle enzyme.
Conclusion: Atorvastatin 20 mg has a dramatic effect on the lipid but moderate effect on CRP. The two different types of Atorvastatin (group A and B) have similar effect on both safety and efficacy.

Keywords: Atorvastatin, LDL, CRP

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Coronary artery disease has become a major health problem in Asia(1). Dyslipidemia with high serum cholesterol (Total cholesterol & LDL) and low HDL were common risk factors(2,3). Manipulation of TC and LDL, preferably with Statin, has been the major contribution in lowering both new incident and recurrent morbidity of cardiovascular diseases(4-7). Atorvastatin are among the most widely used Statins in the world and in Southeast Asia. In addition to lowering serum cholesterol, Atorvastatin lowers CRP, an index of inflammation. There are now generic versions of Atorvastatin available in Thailand making the drug accessible to more population at risk. The authors took on the study to investigate the response of lipid and inflammatory markers to 20 mg Atorvastatin and to compare the two types of Atorvastatin.
Research Design

Subjects

Patients, aged between 20 and 75, with known coronary artery disease. They were informed in detail and full understanding of the research methodology such as the prevention of high cholesterol by atorvastatin complication or adverse effect of the drugs, the difference of drug comparison were eligible for the present study. Their serum total cholesterol had to be more than 200 mg/dl but less than 400 mg/dl and their LDL had to be more than 100 mg/dl but less than 240 mg/dl for which drug treatment was deemed necessary. The exclusion criteria were current therapy with any drug for dyslipidemia, allergic to Atorvastatin, trigly-ceride level more than 400 mg/dl, pregnancy, active or chronic liver disease, life expectancy to be shorter than 6 months for any disease or reason, chronic renal failure with Cr > 2.0 mg/dl, concomitant use of other drugs that might interfere with Atorvastatin or Cholesterol level e.g., fibrate, cyclosporin or other immunosuppressive drug, active ischemia post acute coronary syndrome period, severe myocardial ischemia by non-invasive test or coronary angiography, poor LV systolic function or congestive heart failure requiring treatment with digitalis, diuretic or vasodilators, hemodynamic important valvular heart disease, history of ileal bypass surgery, active inflammatory or connective tissue disease, or unwilling to participate or no consent form.

Material and Method

Prior to randomization, baseline screening blood analysis of individual was done for serum lipid, liver enzyme (SGOT & SGPT), creatinine, muscle enzyme (CPK & LDH), and high sensitivity C-reactive protein (hs-CRP). The DSL-10-42100 ACTIVE US CRP ELISA is an enzymatically amplified “two step” sandwich-type immunoassay. In the assay, standards, controls, and unknown samples were incubated in microtitration wells that had been coated with anti-US CRP antibody. After incubation and washing, the wells were treated with another anti-US CRP detection antibody labeled with the enzyme horseradish peroxidase (HRP). After a second incubation and washing step, the wells were incubated with the substrate tetramethylbenzidine (TMB). An acidic stopping solution was then added and the degree of enzymatic turnover of the substrate was determined by dual wavelength absorbance measurement at 450 and 620 nm. The absorbance measured was directly proportional to the concentration of US CRP present. A set of US CRP standards was used to plot a standard curve of absorbance versus US CRP concentration from which the US CRP concentrations in the unknown can be calculated. If no exclusion criterion was met, the subjects were randomized to receive either group A and Group B.

The medicines were packaged in bottles that looked identical. Each bottle contained 60 tablets of either 20-mg Atorvastatin with instruction to be taken once a day in the evening or before bedtime. The actual tablet of both types of Atorvastatin did not have the same appearance. Each bottle was labeled with a special code for individual patient identification. Once the subject was identified, the investigator using block randomly picked the medicine bottle from the cabinet and that bottle’s code number became the patient’s code number.

Follow-up

All patients (group A and group B) were contacted by phone after 2-4 weeks of treatment asking about any complaint and any possibility of side effect. They were instructed to return to the clinic for a blood test and later to meet with research physicians after 60 days of initial therapy or earlier if there was an adverse effect. After 60 days, they were switched to the other arm of therapy for the second period of 60 days therapy using the same code number. They were instructed to return to the clinic for a blood test and later to meet with research physicians after 120 days of initial therapy or earlier if there was an adverse effect. At baseline, 60 days and 120 days, blood tests were done for lipid profile, liver enzyme, creatinine, muscle enzyme, and hs-CRP. Special test tubes and packages were made available for blood storage. All blood samples were sent to the central laboratory for analysis[21]. The pill count was done at each clinic visit. Both patients and physicians were blind to the type of medication being given. Elevations of liver enzyme (SGOT & SGPT) more than three times or muscle enzyme (CPK or LDH) more than 10 times baseline value were judged abnormal and warranted withdrawal from the therapy. The patients were informed that they could withdraw from the study at anytime.

Study monitoring

Medicine count was used as the measurement of compliance. If total pill count showed more than 20% of medication left untouched (> 12 pills) subjects were discharged from the present study. The principal investigator was responsible for the result monitor. All data were reviewed at two weeks interval. Any
appearance of unusual events or adverse effect were alerted to the investigators of each hospital.

**Study endpoints**

The primary endpoints were the mean absolute and percent reduction of total cholesterol and LDL for the whole cohort and in either group at 60 and 120 days. Secondary endpoints included percent change of mean HDL, Triglyceride and hs-CRP level for the whole cohort and in either group at 60 and 120 days. The number of subjects who withdrew from the present study for clinical side effect or biochemical abnormality was used as tertiary endpoints.

**Statistic analysis**

Results were expressed as means and SD or as proportion (percentage). The Student’s t-test was used for parametric data when normal distribution and equal dispersion were recognized. Differences in the categorical data were analyzed by χ² analysis and the Fisher’s exact test was used when appropriate. Log-scale method was used in place where wide variation of values was noted. P-value of < 0.05 determined the statistical significance.

**Results**

One hundred and ten patients were enrolled in the present study. Among these, 12 were patients with stable angina. The majority of subjects were prone to developing vascular disease and had no symptoms (primary prevention). The mean age was 54 years with the majority (62%) being female. The two groups were similar to all variables examined. Their profiles are shown in Table 1. The starting total cholesterol and LDL were around 250 and 175 mg/dl respectively. The hs-CRP level, however, had a wide range of value in both groups.

Fifty-four patients were assigned to group-A, who took Pfizer Atorvastatin for the first 8 weeks. One patient in group-A withdrew at the first telephone visit without a blood test. Two more patients (one from each group) withdrew after week 8 with one set of follow-up blood tests obtained. One hundred and nine patients and 107 had a blood test at week 8 and week 16 respectively. For the whole cohort the 20 mg of Atorvastatin lowered TC by 83.7 mg/dl or 32% and lowered LDL by 78 mg/dl or 44% at week 8 (Table 2).

At week 8 visit blood test the mean absolute reduction of TC was 86 mg/dl or 33% from baseline for group-A and 81 mg/dl or 31% from baseline for group-B. The mean reduction of LDL was 80 mg/dl (45% from baseline) for group-A and 77 mg/dl (43% from baseline) for group-B. The difference was similar statistically. After switching the drug, subjects in both groups had the lipid reduction pattern maintained at 120 days. The mean absolute reduction of TC (78mg/dl on group B and 85mg/dl on group A) and LDL (72mg/dl on group B and 73 mg/dl on group A) for the second half of the present study were not statistically significantly different. The mean absolute reduction of TC and LDL and mean percentage reduction of TC and LDL from base line at week 8 and at week 16 were not different for the whole cohort and were not different for each group (Table 2).

At week 8, 84.6% of the subjects in group A and 87.7% of subjects in group B achieved more than 30% reduction of LDL with Atorvastatin 20 mg. 59% of population in group A compared to 60% of population group B had their LDL lower to less than 100mg/dl. The numbers were not statistically different. HDL level did not change significantly with either group A or group B and with only 2.5% and 6% increase over 8 weeks of treatment. The percentage change did not differ from week 8 to week 16 after switching the medicine in either subject groups. TG was reduced by group A and by group B to 15.9% and 14.1% respectively without any difference statistically.

There was a wide variation of hs-CRP value in this population with the range of 0.305-97.25 mg/dl (or 305-97250 mcg/dl) for the whole cohort. The wide variation was also noted in each group. Log scale was used to correct for such a wide variation. The baseline Log hs-CRP was 3.79 (0.38) in Group A and 3.86 (0.41) mg in Group B. The Log value hs-CRP at 8 week was 3.75 (0.37) for group A and 3.82 (0.39) for group B. After switching the medicine, the Log value of hs-CRP continued to be lower in both groups with Log value hs-CRP for group A was 3.73 (0.47) and for group B was

**Table 1. Baseline characteristics of subjects**

<table>
<thead>
<tr>
<th></th>
<th>Whole cohort</th>
<th>Group-A</th>
<th>Group-B</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (%)</td>
<td>110</td>
<td>54 (49)</td>
<td>56 (51)</td>
</tr>
<tr>
<td>Male (%)</td>
<td>38</td>
<td>45</td>
<td>29</td>
</tr>
<tr>
<td>Age</td>
<td>54 ± 10</td>
<td>54 ± 11</td>
<td>54 ± 10</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>251 ± 39</td>
<td>251 ± 41</td>
<td>253 ± 39</td>
</tr>
<tr>
<td>HDL</td>
<td>55 ± 15</td>
<td>54 ± 15</td>
<td>55 ± 15</td>
</tr>
<tr>
<td>LDL</td>
<td>174 ± 37</td>
<td>174 ± 39</td>
<td>175 ± 33</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>160 ± 80</td>
<td>168 ± 103</td>
<td>159 ± 79</td>
</tr>
<tr>
<td>CPK</td>
<td>117 ± 86</td>
<td>120 ± 78</td>
<td>114 ± 94</td>
</tr>
<tr>
<td>SGOT</td>
<td>26 ± 11</td>
<td>28 ± 11</td>
<td>25 ± 12</td>
</tr>
</tbody>
</table>

(See abbreviation)
Table 2. Primary endpoints (mean absolute reduction)

<table>
<thead>
<tr>
<th></th>
<th>Whole cohort</th>
<th>Group-A</th>
<th>Group-B</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>P-Atorva first</td>
<td>B-Atorva first</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>n = 54</td>
<td>n = 56</td>
<td></td>
</tr>
<tr>
<td>Mean (SD) TC reduction wk 8</td>
<td>83.7 (42.1)</td>
<td>86.3 (44.7)</td>
<td>81.3 (39.8)</td>
<td>0.55</td>
</tr>
<tr>
<td>Mean (SD) TC reduction wk 16</td>
<td>81.7 (43.8)</td>
<td>85.1 (37.9)</td>
<td>78.0 (49.7)</td>
<td>0.41</td>
</tr>
<tr>
<td>Mean (SD) % TC reduction wk 8</td>
<td>31.9 (16.9)</td>
<td>32.9% (20.2)</td>
<td>31.0% (13.1)</td>
<td>0.56</td>
</tr>
<tr>
<td>Mean (SD) % TC reduction wk 16</td>
<td>31.4 (16.9)</td>
<td>32.8% (12.4)</td>
<td>29.9% (20.8)</td>
<td>0.38</td>
</tr>
<tr>
<td>Mean (SD) LDL reduction wk 8</td>
<td>78.6 (33.6)</td>
<td>80.1 (36.4)</td>
<td>77.1 (31.0)</td>
<td>0.65</td>
</tr>
<tr>
<td>Mean (SD) LDL reduction wk 16</td>
<td>72.7 (40.8)</td>
<td>73.3 (36.4)</td>
<td>71.9 (45.6)</td>
<td>0.87</td>
</tr>
<tr>
<td>Mean (SD) % LDL reduction wk 8</td>
<td>44.0 (14.3)</td>
<td>45.1% (15)</td>
<td>43.1% (13.7)</td>
<td>0.46</td>
</tr>
<tr>
<td>Mean (SD) % LDL reduction wk 16</td>
<td>40.7 (21.6)</td>
<td>40.9% (18.0)</td>
<td>40.5% (25.2)</td>
<td>0.93</td>
</tr>
<tr>
<td>Mean (SD) % HDL change wk 8</td>
<td>3.6 (7.9)</td>
<td>2.5% (13.4)</td>
<td>6.06% (15.8)</td>
<td>0.23</td>
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<tr>
<td>Mean (SD) % HDL change wk 16</td>
<td>1.5 (8.4)</td>
<td>6.35% (18.2)</td>
<td>2.21% (15.3)</td>
<td>0.22</td>
</tr>
<tr>
<td>Mean (SD) % TG change wk 8</td>
<td>33.6 (71.0)</td>
<td>15.9% (32.0)</td>
<td>14.1 (39.1)</td>
<td>0.80</td>
</tr>
</tbody>
</table>

Table 3. High sensitivity-C Reactive Protein (hs-CRP) for the whole cohort and each group at baseline and after 20 mg of Atorvastatin

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>8th week</th>
<th>16th week</th>
<th>p-value A vs. B</th>
<th>p-value log A vs. log B</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Mean ± SD</td>
<td>9424 ± 10194</td>
<td>9058 ± 1227</td>
<td>8860 ± 9975</td>
<td>0.29</td>
</tr>
<tr>
<td>n = 54</td>
<td>Range</td>
<td>305-56590</td>
<td>900-69980</td>
<td>296 ± 56885</td>
<td>3.72 (0.46)</td>
</tr>
<tr>
<td></td>
<td>log ± SD</td>
<td>3.79 ± 0.38</td>
<td>3.75 ± 0.37</td>
<td>3.73 ± 0.47</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>Mean ± SD</td>
<td>11936 ± 15593</td>
<td>10042 ± 10593</td>
<td>10243 ± 22472</td>
<td>0.29</td>
</tr>
<tr>
<td>n = 56</td>
<td>Range</td>
<td>1130-97250</td>
<td>1000-55500</td>
<td>259 ± 151675</td>
<td></td>
</tr>
<tr>
<td></td>
<td>log ± SD</td>
<td>3.83 ± 0.41</td>
<td>3.82 ± 0.39</td>
<td>3.72 ± 0.46</td>
<td></td>
</tr>
<tr>
<td>p-value (A vs. B)</td>
<td>0.29</td>
<td>0.67</td>
<td>0.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>p-value (log A vs. log B)</td>
<td>0.36</td>
<td>0.34</td>
<td>0.89</td>
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</table>

Discussion

As a result of many clinical trials, Statin has been recommended for both secondary and primary prevention of vascular events\(^8\text{–}^{10}\). Cost effectiveness has also been established for both primary and secondary prevention\(^{11}\). Atorvastatin is currently the most widely used statin in the world and in Thailand. Many doses of Atorvastatin were proven to be effective in clinical trials. One study\(^{22}\) (GREACE study) showed that 90% of the patients had their LDL lower by 100 mg/dl (the desired level at that time) after an acute coronary event with the dose of 20 mg of Atorvastatin. ASCOT study\(^{10}\) on Primary prevention using flat dose (empirical) of 10 mg of Atorvastatin in addition to anti-hypertensive drugs showed that mean reduction of LDL was 38 mg/ml or 1 mmol/ml in the 10 mg Atorvastatin group compared to the placebo group. The present study was set out to evaluate the drug responsiveness of Atorvastatin in a selected group of the Thai population. The LDL, on
average, was dropped by 45% or by 78 mg/dl at week 8 with 20 mg of Atorvastatin. It is likely that the Thai population has a good response to Atorvastatin in terms of LDL reduction. However, the population in the present study was composed mostly of people who had never had vascular events i.e., for primary prevention reason. Only 12% had stable angina as the co-existing symptom. Their baseline LDL level however was relatively high. It was interesting to see that the baseline LDL level dictated the responsiveness to Atorvastatin. People with their LDL higher quartile than 1 had an average of absolute 10% additional reduction of LDL level compared to people with relatively low baseline LDL.

Atorvastatin from two different manufacturers were not different in their ability to lower LDL. In the present study, we used the switching method at week 8 and continued the medications for another 8 weeks. At week 16, the LDL levels were not different from week 8 in either group. The LDL levels of both groups were almost the same at week 8 and week 16. This suggested that the drugs from the two manufacturers could be interchangeable without any significant change in LDL level or any adverse effect. The authors observed no significant changes in the level of HDL although there was a minor drop in Triglyceride level. This is in line with other previous Atorvastatin trials. The dropout rate (1 during the initial phrase and 2 after the first visit) were predictable and similar to the previous clinical studies. The biochemical abnormalities noted here were mostly transient with no significant myopathy or liver toxicity. Giving this safety profile, the drug is likely to be prescribed empirically in the future in Thailand.

Atherosclerosis may in part, be an inflammatory disease(16), circulating factors related to inflammation, especially CRP may be predictors of cardiovascular events(17). There was a recommendation from the Centers for Disease Control and Prevention and the American Heart Association that it was reasonable to measure CRP as an adjunct to the measurement of established risk factors in order to assess the risk of coronary heart disease(18). It is still controversial whether to use CRP for such purposes since the level were variable among age, sex, and race(19). Compared to the conventional risk factors assessment, CRP at least in some study was not shown superior in predicting risk(20). CRP level in our study was widely variable. The range from 305 to 97250 mcg was observed because of high sensitivity method being used. Giving this wide variation, the use of hs-CRP as the method of risk evaluation may not be practical. Log-scale method had to be used in our study to correct such wide variation. For the whole cohort, 20 mg of Atorvastatin lowered hs-CRP level by 10%. However, the responses were inhomogeneous i.e. hs-CRP went up in certain individuals. For the purpose of comparison, Atorvastatin from the two manufacturers were similar in term of baseline Log-value CRP and its inhomogeneous response to the drug. Giving these pictures, CRP especially hs-CRP may not be ready to be used as risk factors for vascular events or used as response to therapy in Thai populations.

**Conclusion**

Atorvastatin of 20 mg lowered LDL by 45% and hs-CRP by 10% in Thai subjects with relatively high LDL level, majority of which did not have pre-existing vascular disease. The drug was quite safe with a low dropout rate and only with transient reversible biochemical abnormality. Atorvastatin group B performed as well for such purposes as Atorvastatin group A.

**Abbreviation**

LDL = low density lipoprotein  
HDL = high density lipoprotein  
TG = triglycerides  
TC = total cholesterol  
Cr = creatinine  
SGOT = serum glutamic oxaloacetic transaminase  
SGPT = serum glutamic-pyruvic transaminase  
CPK = creatin phosphokinase  
LFT = liver function tests  
hs-CRP = high-sensitivity C-reactive protein  
B = Berlin-atorvastatin  
P = Pfizer-atorvastatin  
ELISA = enzyme-linked immuno sorbent assay  
Group A = Pfizer-atorvastatin  
Group B = Berlin-atorvastatin  
mg = milligram  
mcg = microgram

**References**


ผลตอบสนองของไขมัน แอลดีแอล และ ซีอาพี ต่อยาอะทอสะเตติน ในคนไทยกลุ่มหนึ่ง

สรณ บุญใบชัยพฤกษ์, สายัณห์ ชีพอุดมวิทย์, ประดิษฐ์ ปัญจวีนิน, ถาวร สุทธิไชยากร, ธนวัฒน์ เบญจานุวัตร, บัญชา สุขอนันต์ชัย, บุญใบชัยพฤกษ์

ภูมิหลัง: ระดับไขมันแอลดีแอล และ ซีอาพี ในเลือดเป็นปัจจัยเสี่ยงสำคัญต่อการเกิดโรคหัวใจและหลอดเลือด ยาในกลุ่มสะเตตินสามารถปรับลดระดับไขมันแอลดีแอล และ ซีอาพี

วัตถุประสงค์: เพื่อตรวจสอบระดับ ชื่อยา ในคนไทยกลุ่มหนึ่งที่มีความจำเป็นต้องใช้สะเตติน และเพื่อตรวจสอบผลของยาอะทอสะเตตินต่อระดับไขมันแอลดีแอลและซีอาพี และ เพื่อตรวจสอบประสิทธิภาพและความปลอดภัยของยาอะทอสะเตตินสองชนิด

วัสดุและวิธีการ: คณะผู้นิพนธ์ได้ทำการสุ่มเลือกผู้ที่มีข้อบ่งชี้ว่าจะได้รับประโยชน์จากการรักษาด้วยสะเตตินให้ได้รับยาอะทอสะเตติน ขนาด 20 มิลลิกรัมโดยแบ่งออกเป็นสองกลุ่ม 8 สัปดาห์กลุ่มแรกจะได้รับยาอะทอสะเตตินที่ผลิตจากไฟเซอร์ และกลุ่มที่สองจะได้รับยาอะทอสะเตตินที่ผลิตจากเบอร์ลิน โดยมีการสลับกลุ่มหลังจากนั้น และได้ทำการรักษาต่อไปอีก 8 สัปดาห์ ผู้มีโรคที่ต้องการติดตามอาการและความผิดปกติทางคลินิกตลอดเวลาการศึกษา และได้มีการตรวจเลือดระดับไขมันแอลดีแอล ซีอาพี ค่าการทำงานของตับ และเอนไซม์กล้ามเนื้อ กล้านิวเคลียร์ เพื่อเปรียบเทียบกับหลังจากที่ได้รับยาในสัปดาห์ที่ 8 และ สัปดาห์ที่ 16

ผลการศึกษา: มีผู้ป่วย 110 คนเข้าร่วมการศึกษา โดยที่ 54 คนได้รับยาอะทอสะเตตินที่ผลิตจากไฟเซอร์ใน 8 สัปดาห์แรกที่ผลิตโดยไฟเซอร์ ไอ ayatailai ของผู้ป่วย 54 ปี โดยที่ร้อยละ 38 คนมี效益เป็นราย ระดับไขมันในเลือดต่ำลงคือ คอเลสเตอรอล 251 แอลดีแอล 174 เฮชดีแอล 55 และไตรกลีเซอร์ไลด์ 160 มิลลิกรัมต่อเดซิลิตร พบว่าระดับซีอาพีมีเกิดขึ้นที่จำนวนมาก และต้องใช้ค่า logscale ในการประเมินแทนค่าเฉลี่ย 107 คนเข้าร่วมการวิจัยจนจบ 16 สัปดาห์ และพบว่าระดับซีอาพีลดลงในเวลา 20 มิลลิกรัมสามารถลดระดับ คอเลสเตอรอล 32%, แอลดีแอล 44% และระดับซีอาพี 10% โดยที่ยาอะทอสะเตตินที่ผลิตจากไฟเซอร์ มีผลลดระดับ คอเลสเตอรอล, แอลดีแอล และซีอาพีเป็นมากกว่าการใช้ซีอาพี ฟิน นอกจากนี้แล้ว ที่สำคัญคือมีผลเป็นประจำต่อการชื่นชอบ และผลกระทบต่อการทำงานของตับ และเอนไซม์กล้ามเนื้อ

สรุป: ยาอะทอสะเตตินขนาด 20 มิลลิกรัม สามารถลดระดับไขมันคอเลสเตอรอลได้เป็นอย่างดี ในกลุ่มคนไทยที่เจาะเกี่ยวกับการศึกษา ค่าชื่อยาที่คนไทยที่อยู่ในภาวะดีได้รับผลที่ดีมาก และยาอะทอสะเตตินมีผลเป็นจำนวนมากในการลดระดับซีอาพีในกลุ่มนี้ ยาอะทอสะเตตินจากไฟเซอร์ และจากเบอร์ลินไทยให้ผลเหมือนกันในเรื่องประสิทธิภายนอกและความปลอดภัย