Hypercholesterolaemia is a potent cardiovascular (CV) risk factor. In SA, white Afrikaner, Jewish and Asian populations have one of the highest familial hypercholesterolaemia (FH) carrier rates in the world.

Studies have shown that only 10.6% of people with hypercholesterolaemia receive a statin or treatment. There is a within-person variability of cholesterol measurements and therefore a one-time measurement of cholesterol tends to underestimate the strength of the relationship between cholesterol and coronary heart disease.

Multiple cholesterol measurements over time (decades) such as done in some epidemiological studies are therefore important to get an idea of life-time risk of atherosclerosis as the disease develops over decades.

An example of such a study is the Framingham Heart Study that extended the results of the Johns Hopkins study. In this study 1478 adults without incident CVD were recruited from the Framingham Offspring Study at the age of 55 years if they were free from CVD and they were then followed for 20 years for the occurrence of CVD.

When they were selected their cholesterol levels were evaluated as it was done on a number occasions in the past. The authors were thus able to tract the cholesterol levels for decades before the people were 55 years old and from 55 onwards they were followed for CVD events.

CONCLUSIONS

1. Cumulative exposure to hyperlipidaemia in young adulthood increases the risk of CHD later and they may benefit from treatment.
2. Current guidelines base risk assessment on a 1-time measurement of cholesterol and other risk factors. This practice will necessarily exclude many young adults from intensive treatment.
3. Akin to smoking pack-years we would multiply the cholesterol levels with the duration of exposure to elevated cholesterol and so have a LDL-years measurement to use in risk assessments.
4. The most important question now is when to start treatment and how young should we start treatment. Is sooner the better the answer?

References available on request.

Adding fenofibrate to statin therapy in patients with mixed dyslipidaemias

• Fenofibrate plus simvastatin therapy significantly improved ALL lipoprotein abnormalities vs simvastatin alone (p < 0.001)²

Change from baseline to 12 weeks in lipid parameters²

<table>
<thead>
<tr>
<th></th>
<th>Fenofibrate + Simvastatin</th>
<th>Simvastatin</th>
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</thead>
<tbody>
<tr>
<td>TG</td>
<td>-35.1</td>
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<tr>
<td>TC</td>
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<td>+23.5</td>
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<tr>
<td>LDL-C</td>
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<td>HDL-C</td>
<td>+9.7</td>
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<tr>
<td>non-HDL-C</td>
<td>-38.0</td>
<td>+57.0</td>
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<tr>
<td>Apo B</td>
<td>+2.0</td>
<td>-22.0</td>
</tr>
</tbody>
</table>

*All percent changes from baseline were statistically significant (p<0.016)²


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