The Health Research Society of Canterbury

Call for Abstracts for the

2013 Grand Round Series

Rolleston Lecture Theatre, Ground Floor,
University of Otago, Christchurch,

First meeting: **Friday 3th May, 12:30 to 13:30.**
Second meeting: **Friday 10th May 12:30 to 13:30.**

Closing Date for abstracts: Monday, 15th April

Selected abstracts will be presented as 10 minute oral presentations with a few minutes for questions. A total of 8 abstracts will be selected, 4 for each session. Abstracts will be published in **The New Zealand Medical Journal.** See following page for **Abstract Details**. Abstracts not in the correct format cannot be accepted for consideration.

Prizes for the best student presentation during the year will be made at the AGM at the end of the year (date to be finalized)

Abstracts should be submitted to the Society by email to
tracy.melzer@nzbri.org
Place the words "**HRSC abstract**" in the subject line.

Background: The Health Research Society (HRSC) was formed in 1971 (originally named the Christchurch Medical Research Society, CMRS). Its role is to provide a forum for the presentation of health research and to promote such research generally. Membership is open to all people either involved in or simply interested in health research, and is available for an annual fee of $10 payable to the treasurer. For more information on the Society, its activities, and how to be involved, go to the web site http://www.hrsc.org.nz.

Contacts for 2013

Chairperson: A/Prof Steven Gieseg, School Biological Sciences, University of Canterbury,
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Treasurer: A/Prof Nick Draper, School of Sport and Physical Education, University of Canterbury,
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Abstract format for the HRCS abstracts to be published in the New Zealand Medical Journal.

Abstracts should be submitted as MS word files with the page margins set to 2.5 cm right and left hand side. The abstracts are to be written in Font Times Roman 12pt, 1.5 spacing and no longer than 300 words excluding title, authors names and affiliations, all which should be **bolded**. The title should be in sentence case and no longer than 150 characters. The list of authors should begin on the same line and the name of the presenting author underlined. The names of the authors should be in the format of initials followed by the surname. Each author's name is to be separated by a comma. Affiliations of each author are to be indicated by a superscript number after the surname. There should be a line gap between the abstract text and the title and authors. There should be no more than 3 references given. See the below example.

The macrophage derived 7,8-dihydroneopterin preventions oxLDL formation,

SP Gieseg[1], J Roake[2], [1]Free Radical Biochemistry Laboratory, School of Biological Sciences, University of Canterbury, Christchurch, [2]Department of Surgery, Christchurch Hospital, Christchurch.

Introduction: Plasma neopterin is an excellent marker of inflammation and is found in elevated levels in patients with cardiovascular disease (1). Neopterin is the oxidation product of 7,8-dihydroneopterin, which is secreted by human macrophages when stimulated with γ-interferon during inflammation. The purpose for this cellular response is not known but we have previously shown 7,8-dihydroneopterin to be a potent antioxidant able to protect macrophages from oxidative stress (1). The death of macrophages by oxidised low density lipoprotein (OxLDL) is a key driver in the development of complex plaque within the artery wall. We examined whether 7,8-dihydroneopterin can inhibit oxLDL mediated macrophage cell death and whether this oxidant was formed in atherosclerotic plaques.

Methods: Human monocytes isolated from whole blood were differentiated into macrophages over 14 days. OxLDL was prepared by copper oxidation of LDL purified by ultracentrifugation. Plaques supplied from carotid endarterectomy surgery were analysed for total neopterin by HPLC.

Results and Discussion: 7,8-Dihydroneopterin inhibited oxLDL induced necrosis in macrophage cells in a dose dependent manner. The rapid loss of glutathione by oxLDL was inhibited, as was the formation of intracellular oxidants. Neither phosphatidylserine exposure nor cytochrome c release was inhibited by the 7,8-dihydroneopterin treatment but oxLDL uptake was significantly reduced. Neopterin was detected in all plaques analysis at concentrations capable of inhibiting oxLDL formation. Considering the advanced stage of the plaques analysed, this data suggests 7,8-dihydroneopterin may be a significant factor controlling plaque stability and growth.

1) Gieseg, S. P. *et al*. (2007) British Journal of Pharmacology, v153, 627-635.