

DIABETES RESEARCH AND CLINICAL PRACTICE

XIII CONGRESS OF THE INTERNATIONAL DIABETES FEDERATION

Hosted by Diabetes Australia



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ABSTRACTS



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INTRODUCTION

This book contains the submitted abstracts for the oral and poster free communications accepted for presentation at the 13th Congress of the International Diabetes Federation. The abstracts have been reproduced in the form in which they were submitted.

Abstracts for plenary lectures and symposia are not included. The manuscripts of these presentations will be published in the Proceedings of the Congress which may be ordered at the Elsevier desk at the Congress.

The book is arranged with the oral free communications at the front followed by the poster free communications. The abstracts are arranged and indexed according to the session in which they are presented. For example, ORA-015-003 refers to the third presentation in session of 15 of the oral free communications and POS-003-172 refers to poster 172 in the third poster session. The poster presentation number also represents the poster board number, ie POS-003-172 will appear on poster board No. 172. For convenience, a preliminary programme is included in this book. The final programme, which may contain some minor alterations, is included in the programme booklet in your satchel.

Programme and Publications Committees
13th IDF Congress

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POS-002-024

ISOPROTERENOL DECREASES ACTIVATION BY INSULIN OF INSULIN RECEPTOR KINASE IN INTACT RAT ADIPOCYTES.

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It has been previously shown that β -adrenergic agonists produce insulin(I)-resistance both in vivo and in vitro. We have studied the effect of isoproterenol(ISO) on I-binding and activation of insulin receptor kinase(IRK) in intact rat adipocytes. Cell concentration was 2×10^5 /ml in buffer containing 2%BSA and 40mM Hepes (pH 7.4). Preincubation with ISO (10 μ M) was for 30min at 37°C. ¹²⁵I-I was then added to a fraction of the cells for 15min at 37°C to measure I-binding. The other cells were exposed to various concentrations of I for 15min at 37°C in order to activate the cellular IRK. IRK was then isolated and immunoprecipitated in the presence of phosphatase inhibitors. Under these conditions, IRK-activity that resulted from exposure to insulin while the cells were intact, was preserved. It was subsequently measured in the presence of 0.5 μ M ³²P-ATP and 1mg/ml histone and was (fmol P into histone/pmol I-binding activity/min):

Insulin(ng/ml):	0	4	20	500
-ISO :	4.6 \pm 1.0	30.5 \pm 5.1	93.8 \pm 11.6	124.8 \pm 39.3
+ISO :	4.3 \pm 1.1	22.8 \pm 2.5	66.1 \pm 1.0	112.0 \pm 27.8

Mean values for trace I-binding to the cells were: -ISO:0,49 \pm 0.12; +ISO:0,45 \pm 0.08%.

Conclusions: (1)Exposure of the cells to insulin stimulated IRK-activity 25-30fold. (2)Activation of IRK by I in intact cells was reduced up to 30% in the presence of ISO. (3)Only a minimal, if any, reduction of I-binding to the cells was observed in the presence of ISO. (4)Our data thus suggest that ISO reduces I-activation of IRK in intact cells on a post-binding level.