

# Ischemia-Responsive Protein (irp94) Is Up-Regulated by Endoplasmic Reticulum Stress

Seung-Whan Kim<sup>a</sup>, In-Sool Yoo<sup>a</sup>, Hyeon-Song  
Koh<sup>b</sup> and O-Yu Kwon<sup>c,\*</sup>

<sup>a</sup> Department of Emergency Medicine, Chungnam  
National University Hospital, Taejon 301-040, Korea

<sup>b</sup> Department of Neurosurgery, Chungnam National  
University Hospital, Taejon 301-040, Korea

<sup>c</sup> Department of Anatomy, College of Medicine,  
Chungnam National University, Taejon 301-747,  
Korea. Fax: 82-42-586-4800.  
E-mail: oykwon@cnu.ac.kr

\* Author for correspondence and reprint requests

Z. Naturforsch. **56c**, 1169-1171 (2001);  
received September 9/October 22, 2001

Ischemia Responsive Protein (irp94), Endoplasmic  
Reticulum (ER), FRTL-5 Cells

The expression of the ischemia-responsive protein (irp94) was enhanced by endoplasmic reticulum (ER) stress inducing drugs such as brefeldin A (BFA), calcium ionophor A23187, dithiothreitol (DTT) and tunicamycin in fisher rat thyroid epithelial cell line (FRTL-5 cells). In particular, irp94 mRNA expression was increased dose dependently by tunicamycin, and there was increased irp94 expression when the cells were incubated with the thyroid-stimulating hormone (TSH) together.