Deferoxamine Induces Endoplasmic Reticulum Stress in PC12 Cells Young-Bum Yoo^a, Kyeong Ryong Lee^b,

Seung-Whan Kim^c, Kisang Kwon^d, Tae-Won Goo^e, and O-Yu Kwon^{d,*}

Taeion 301-747. Korea

- ^a Department of Surgery, College of Medicine, Konkuk University, Seoul 143-729, Korea
 ^b Department of Emergency Medicine, College of Medicine, Konkuk University, Seoul 143-729, Korea
 ^c Department of Emergency Medicine, Chungnam National University, College of Medicine,
- d Department of Anatomy, Chungnam National University, College of Medicine, Taejon 301-747, Korea. Fax: +82-42-5 86-48 00.
 E-mail: oykwon@cnu.ac.kr
 e Department of Agricultural Biology, National
- Institute of Agricultural Science and Technology, RDA, Suwon 441-100, Korea
- * Author for correspondence and reprint requests
- Z. Naturforsch. **63 c**, 308–310 (2008); received December 28, 2007

Deferoxamine (DFA, N'-[5-(acetyl-hydroxy-amino)-pentyl]-N-[5-[3-(5-aminopentyl-hydroxy-carbamoyl) propanoylamino]pentyl]-N-hydroxy-butane diamide) is a chelating agent used to remove excess iron from the body and to reduce organ and tissue damage. DFA enhances both iron regulatory protein 1 (IRP1) expression and its endoplasmic reticulum (ER) membrane-binding activity, as occurs in hypoxia, an ER stress, in cultured cells. Here, we show that DFA promotes ER stress via an ER signal pathway.

Key words: Deferoxamine (DFA), Endoplasmic Reticulum (ER) Stress