

Possible misconception

I found out the late DEC-1 induction by cisplatin (48h after treatment), which seemed to contradict the previous reports on early DEC-1 induction by various stimuli [1-4]. After reading the reports carefully, I believe my result is in agreement with them. The seeming contradiction is perhaps due to a misconception to think of Stra 13/DEC-1 as an early response gene (ERG).

ERGs

Early response genes (ERGs) are genes which are activated transiently and rapidly in response to a wide variety of cellular stimuli. Unlike other genes, ERGs are often activated at the transcription level in first round response to stimuli, before any new proteins are synthesized. ERGs are represented by *c-fos*, *c-myc* and *c-jun*.

The techniques to determine an ERG are either PCR plus cycloheximide (a chemical agent that inhibit protein synthesis), or classic methods to assess transcriptional initiation such as luciferase reporter assay, Nucleic Run-on, *in vitro*- transcription *etc.*

About previous reports

All the previous reports [1-4] used either RT-PCR or Northern blots only, without the involvement of cycloheximide treatment. Moreover, no classic methods for transcriptional initiation had been used.

Stra 13/ DEC-1 can be induced as early as 2hrs by RA [1] or 3hrs by cisplatin [4] in certain cell lines. However, hypoxia has been reported to trigger induction of DEC-1 48hrs after the treatment [3]. Since no solid data have been presented so far to suggest Stra 13/DEC-1 an ERG, the late induction of DEC-1 by cisplatin in HCT116 and p53^{-/-} cells could be possible. This is support by the observation of DEC-1 induction 24h after 5-FU treatment.

Why DEC-1 induction appear at 6hrs?

I have checked all my data and found out that the only result showing the early induction was done last December. I didn't work independently at that time and the normalization of GAPDH was actually done by two persons. The different criteria of GAPDH normalization may cause an artifact.

1. Sun, H. and R. Taneja, *Stral3 expression is associated with growth arrest and represses transcription through histone deacetylase (HDAC)-dependent and HDAC-independent mechanisms*. Proc Natl Acad Sci U S A, 2000. **97**(8): p. 4058-63.
2. Ivanova, A.V., et al., *Regulation of STRA13 by the von Hippel-Lindau tumor suppressor protein, hypoxia, and the UBC9/ubiquitin proteasome degradation pathway*. J Biol Chem, 2001. **276**(18): p. 15306-15.
3. Yun, Z., et al., *Inhibition of PPAR gamma 2 gene expression by the HIF-1-regulated gene DEC1/Stral3: a mechanism for regulation of adipogenesis by hypoxia*. Dev Cell, 2002. **2**(3): p. 331-41.
4. Thin, T.H., et al., *Stral3 is induced by genotoxic stress and regulates ionizing-radiation-induced apoptosis*. EMBO Rep, 2007. **8**(4): p. 401-7.