

**CURRICULUM VITAE**  
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**Education Background:**

- 1992-1998: University of Texas Southwestern Medical Center at Dallas  
Ph.D. in Biochemistry and Molecular Biology
- 1986-1990: National Yang-Ming Medical University, Taipei, Taiwan  
B.S. in Medical Technology

**Professional Experience:**

- 2013-present Associate Research Fellow  
Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan
- 2006-2012: Assistant Research Fellow  
Institute of Biomedical Sciences, Academia Sinica, Taipei, Taiwan
- 2005-2006: Scientist  
Product of Development, CellPoint Diagnostics, Inc. USA
- 1999-2005: Postdoctoral Research Fellow  
Program of Molecular Medicine, U. of Massachusetts Medical School
- 1990-1992: Research Assistant  
Academia Sinica, Taipei, Taiwan

**Awards and Honors**

- 1997 Robert A. Welch Predoctoral Fellowship, USA
- 1998 Robert A. Welch Postdoctoral Fellowship, USA
- 2001-2003 Charles King Trust Postdoctoral Fellowship from the Medical Foundation,  
USA

**Patents**

U.S. patterns:

1. Fuchs M., Toner M., **Huang YS.**, Krueger NK. and Wang YX. Devices and methods for enrichment and alteration of circulating tumor cells and other particles (2007).  
*US20070026415*
2. Fuchs M., Wang YX. and **Huang YS.** Devices and methods for enrichment and alteration of circulating tumor cells and other particles (2007). *US20070026469*
3. Fuchs M., Wang YX. and **Huang YS.** Devices and methods for enrichment and alteration of circulating tumor cells and other particles (2007). *US20070026419*
4. Fuchs M., Toner M., Wang YX., **Huang YS.**, Krueger NK. and Huang LR. Devices and methods for enrichment and alteration of circulating tumor cells and other particles (2007).  
*US20070026418*
5. Fuchs M., Toner M., **Huang YS.**, Krueger NK. and Haber DA. Devices and methods for enrichment and alteration of circulating tumor cells and other particles (2007).  
*US20070099207*

**Publications:**

1. Chen CY, Chen YT, Wang JY, **Huang YS**, Tai CY (2016) Postsynaptic Y654 dephosphorylation of  $\beta$ -catenin modulates presynaptic vesicle turnover through increased n-cadherin-mediated transsynaptic signaling. *Dev Neurobiol.* [Epub ahead of print]

2. Tsai LY, Chang YW, Lee MC, Chang YC, Hwang PI, **Huang YS\***, Cheng CF\* (2016) Biphasic and stage-associated expression of CPEB4 in hepatocellular carcinoma. *PLoS ONE* 11:e0155025 (\*correspondence)
3. Fong SW, Lin HC, Wu MF, Chen CC, **Huang YS\*** (2016) CPEB3 deficiency elevates TRPV1 expression in dorsal root ganglia neurons to potentiate thermosensation. *PLoS ONE* 11:e0148491
4. Wang KC, Tang SC, Lee JE, Li YI, **Huang YS**, Yang WS, Jeng JS, Arumugam TV, Tu YK (2016) Cerebrospinal fluid high mobility group box 1 is associated with neuronal death in subarachnoid hemorrhage. *J Cereb Blood Flow Metab.* [Epub ahead of print]
5. Chen PJ, Weng JY, Hsu PH, Shew JY, **Huang YS\***, Lee WH\* (2015) NPGPx modulates CPEB2-controlled HIF-1 $\alpha$  RNA translation in response to oxidative stress. *Nucleic Acids Research* 43:9393-404 (\*correspondence)
6. Huang WH, Chao HW, Tsai LY, Chung MH and **Huang YS\*** (2014) Elevated activation of CaMKII $\alpha$  in the CPEB3-knockout hippocampus impairs a specific form of NMDAR-dependent synaptic depotentiation. *Frontiers in Cellular Neuroscience* 8: e367
7. Chang YW and **Huang YS\*** (2014) Arsenite-activated JNK signaling enhances CPEB4-Vinexin interaction to facilitate stress granule assembly and cell survival. *PLoS ONE* 9: e107961
8. Chen YC, Sargsyan K, Wright J, **Huang YS\***, Lim C\* (2014) Identifying RNA-binding residues based on evolutionary conserved structural and energetic features. *Nucleic Acids Research* 42:e15 (\*correspondence)
9. Tsai LY, Chang YW, Lin PY, Chou HJ, Liu TJ, Lee PT, Huang WH, Tsou YL, **Huang YS\*** (2013) CPEB4 knockout mice exhibit normal hippocampus-related synaptic plasticity and memory. *PLoS ONE* 8: e84978.
10. Chao HW, Tsai LY, Lu YL, Lin PY, Huang WH, Chou HJ, Lu WH, Lin HC, Lee PT, **Huang YS\*** (2013) Deletion of CPEB3 enhances hippocampus-dependent memory via increasing expressions of PSD95 and NMDA receptors. *The Journal of Neuroscience* 33:17008-17022 (cover image)
11. Tang SC, Wang YC, Li YI, Lin HC, Manzanero S, Hsieh YH, Phipps S, Hu CJ, Chiou HY, **Huang YS**, Yang WS, Mattson MP, Arumugam TV, Jeng JS (2013) Functional role of soluble receptor for advanced glycation end products in stroke. *Arterioscler. Thromb Vasc. Biol.* 33:585-94
12. Chao HW, Lai YT, Lu YL, Lin C, Mai W and **Huang YS\*** (2012) NMDAR signaling facilitates the IPO5-mediated nuclear import of CPEB3. *Nucleic Acids Research* 40:8484-98
13. Wang CF and **Huang YS\*** (2012) Calpain 2 activated through the NMDA receptor signaling cleaves CPEB3 and abrogates CPEB3-repressed translation in neurons. *Molecular and Cellular Biology* 32: 3321-3332
14. Chen PJ and **Huang YS\*** (2012) CPEB2-eEF2 interaction impedes HIF-1 $\alpha$  RNA translation. *EMBO Journal* 31: 959-971
15. Peng SC, Lai YT, Huang HY, Huang HD and **Huang YS\*** (2010) A novel role of CPEB3 in regulating EGFR gene transcription via association with Stat5b in neurons. *Nucleic Acids Research* 38: 7446-57
16. Lin AC, Tan CL, Lin CL, Strochlic L, **Huang YS**, Richter JD and Holt CE (2009) Cytoplasmic polyadenylation and CPE-dependent mRNA regulation are involved in *Xenopus* retinal axon development. *Neural Development* 4:8
17. **Huang YS\*** and Richter JD (2007) Analysis of mRNA translation in cultured hippocampal neurons. *Methods in Enzymology* 431:143-62 (\*correspondence)
18. **Huang YS**, Kan MC, Lin CL and Richter JD (2006) CPEB3 and CPEB4 in neurons: analysis of RNA-binding specificity and translational control of AMPA receptor GluR2 mRNA. *EMBO Journal* 25: 4865-4876

## **CPEB2-controlled translation and oxidative stress**

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Cytoplasmic polyadenylation element binding protein 2 (CPEB2) is an RNA-binding protein and translational regulator. CPEB2 can interact with the elongation factor, eEF2, to reduce eEF2/ribosome-triggered GTP hydrolysis *in vitro* and slow down peptide elongation of CPEB2-bound RNA *in vivo*. The interaction of CPEB2 with eEF2 downregulates HIF-1 $\alpha$  RNA translation at elongation under normoxic conditions; however, when cells encounter oxidative stress, CPEB2 dissociates from HIF-1 $\alpha$  RNA, leading to rapid synthesis of HIF-1 $\alpha$  for hypoxic adaptation (Chen et al, 2012, *EMBO J.* 31: 959). The redox-sensitive RNA-binding ability of CPEB2 is later identified to be modulated by nonselenocysteine-containing phospholipid hydroperoxide glutathione peroxidase (NPGPx). NPGPx forms a disulfide bond with the N-terminal Cys157 of CPEB2, which promotes the proper rearrangement of intramolecular disulfide bonds of CPEB2 to enhance its RNA-binding ability and hence downregulate HIF-1 $\alpha$  RNA translation under normoxia. High oxidative stress disrupts the disulfide bonding between NPGPx and CPEB2 that weakens the association of CPEB2 and HIF-1 $\alpha$  RNA to increase HIF-1 $\alpha$  RNA translation (Chen et al, 2015 *NAR* 43: 9394). To investigate whether CPEB2-controlled translation affects oxidative homeostasis *in vivo*, we have generated CPEB2 knockout (KO) mice. CPEB2 KO mice are born alive but most die within 3 days after birth showing no overt defects in anatomy. Whether the cause of death is in part due to altered oxidative stress response is under investigation.