



# Hong Kong Society of Endocrinology, Metabolism and Reproduction

## **Guidelines for Registration & Abstract Submission**

(Forms and instruction guidelines can be downloaded from [www.endocrine-hk.org](http://www.endocrine-hk.org))

### **Registration**

- (1) The HKSEMR Annual Scientific Meeting is open to all members and non-members. Advance registration is required and should be done before 12 Nov 2010.
- (2) Registration is free and registration form should be addressed to Dr Annette Tso by email to [hksemr@gmail.com](mailto:hksemr@gmail.com) or fax to 2816-2863

### **Abstract Submission**

- (1) Submission of abstracts on both basic research and clinical research in the fields of endocrinology, metabolism and reproduction for presentation at the meeting are welcome. Once the abstract is accepted, the presenter will be invited for either a 15 minutes oral presentation or a poster presentation.
- (2) For those who would like to be considered for the presentation awards, a mini-paper (maximum of two A4 pages) describing the major findings of the presentation should be submitted with the abstract. Accepted abstracts and mini-papers will be published in the Program & Abstract Book of the Annual Scientific Meeting.
- (3) Please indicate at the time of submission the following information: (i) name of presenter, (ii) email address, (iii) preference for oral or poster presentation, and (iv) participation in award competition.
- (4) Abstracts and mini-papers for award competition should be addressed to Dr Annette Tso by email to [hksemr@gmail.com](mailto:hksemr@gmail.com). **The deadline for abstract submission is 10 October 2010.** Notification of acceptance will be sent to the presenting author after 8 November 2010 with the instructions for oral and poster preparation.

### **Format of Abstract Preparation**

- (1) Abstract should be typed and justified on both sides using Times New Roman with a font size of 12. The abstract should be typed within the box (15 cm × 20 cm) provided in the abstract submission form. The margins for page setup in A4 paper in Microsoft Word format are: Top 5 cm, Bottom 5 cm, Left 3 cm, Right 3 cm, Gutter 0 cm, Header 0 cm, and Footer 0 cm.
- (2) The title of the abstract should be in bold capital letters, name of the authors and institution/affiliation should be in bold, italic, title case. The main text of the abstract (including acknowledgment of funding support) in capital and small letters. (See sample abstract attached for details.)

### **Format of Mini-Paper Preparation** (for award competition only)

- (1) Mini-Paper is limited to two A4 pages and should be typed in single-column or two-column format and justified on both sides using Times New Roman with a font size of 10. The abstract should be typed within the box (14 cm × 23 cm) provided in the abstract submission form. The margins for page setup in A4 paper in Microsoft Word format are: Top 2 cm, Bottom 3 cm, Left 2 cm, Right 2 cm, Gutter 0 cm, Header 0 cm, and Footer 0 cm.
- (2) The title of the mini-paper should be bold typed in title case. The name of authors, institution/affiliation, and the main text of the paper (including figure legends & acknowledgements) should be typed in capital and small letters. References should be in the format as shown in the attached sample for mini-papers.
- (3) Standard abbreviations are allowed and special abbreviation(s) should be defined when first introduced. The mini-paper will be published in the Program & Abstract Book in black and white only and no color plates will be entertained.
- (4) Should you have further questions regarding the submission of mini-paper, please contact Dr Annette Tso by email at [hksemr@gmail.com](mailto:hksemr@gmail.com).

**GRASS CARP CREB: MOLECULAR CLONING, CHARACTERIZATION,  
AND REGULATION OF TRANSCRIPT EXPRESSION BY  
SOMATOSTATIN AT THE PITUITARY LEVEL**

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CREB, a ubiquitous transcription factor, mediates the stimulatory effects of the AC/cAMP/ PKA pathway on gene transcription by binding directly to the CRE site(s) in the target gene promoter. Although functional CRE sites have been identified in the 5' promoter of growth hormone (GH) gene, pituitary expression of CREB and its role in neuroendocrine control of GH synthesis have not been previously examined. In this study, using grass carp as an animal model, we tested the hypothesis that somatostatin (SRIF), a GH-release inhibitor, could suppress GH gene expression by modulation of CREB expression at the pituitary cell level. As a first step, molecular cloning was performed to establish the structural identity of grass carp CREB using nested PCR coupled to 5'/3' RACE. The full-length cDNA obtained reveals that the a.a. sequence of carp CREB (324 a.a.) is highly homologous (86-90%) to the mammalian counterparts. Functional expression of CREB has confirmed that the newly cloned cDNA encodes a functional protein that can bind to canonical CRE and transactivate gene promoter with tandem repeats of CRE sites. In primary cultures of grass carp pituitary cells, SRIF treatment dose-dependently suppressed cAMP production and GH mRNA level with a concurrent rise in "steady-state" CREB mRNA expression. This increase in CREB mRNA levels could be partly attributed to the enhancement of transcript stability for CREB mRNA after SRIF treatment. The stimulatory effect of SRIF on CREB mRNA expression was mimicked by the AC inhibitor MDL12330A and PKA inhibitor H89. The AC activator forskolin or the cAMP analog CPT-cAMP, in contrast, was effective in reducing the basal expression of CREB mRNA in carp pituitary cells. In parallel experiments with GH3 cells transfected with a reporter construct carrying the 5' promoter of grass carp GH gene, over-expression of carp CREB was found to be inhibitory to basal GH promoter activity. Using 5' deletion analysis, the CREB responsive sequence could be mapped to the region between -742 to -646 of the grass carp GH promoter. These results, as a whole, suggest that SRIF, by inhibiting the AC/cAMP/PKA pathway, can up-regulate CREB gene expression at the pituitary level to inhibit GH gene expression in fish models.

## Effects of Norepinephrine on Growth Hormone Release from the Pituitary of Goldfish, *Carassius auratus*.

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### Introduction

Unlike mammals, teleost fishes are unique for the lack of a hypophyseal portal blood system and their anterior pituitaries are directly innervated by hypothalamic neurons (1). In the goldfish, norepinephrine (NE) fibers have been identified in the basal hypothalamus as well as in the preoptico-infundibular neuronal pathway (2), suggesting that NE may directly influence the pituitary functions.

In this study, we examined the effects of NE on growth hormone (GH) release from the goldfish pituitary under column perfusion. To establish the physiological relevance of NE actions, the interactions between NE and two GH-releasing factors in the goldfish, gonadotropin-releasing hormone (GnRH) and dopamine (DA), were also investigated.

### Materials and Methods

Dispersed goldfish pituitary cells were prepared as described by Chang et al (3) and perfused using the ACUSYS T-S perfusion system. In the studies with GnRH and DA, pituitary fragments were used instead of pituitary cells. GH contents in perfusate fractions were assayed using a goldfish GH radioimmunoassay (4). Data were transformed into "% pretreatment" as defined previously (5) and subjected to statistical analysis using ANOVA followed by Fisher's LSD test ( $P < 0.05$ ).

### Results

Increasing doses of NE (10 nM - 10  $\mu$ M) suppressed basal GH release from perfused goldfish pituitary cells in a dose-dependent manner (Fig.1). Furthermore, a rebound of GH release was observed after the termination of NE treatment. GnRH (50 nM) and DA (0.5  $\mu$ M) consistently stimulated GH release from perfused goldfish pituitary fragments (Fig.2). However, these stimulatory responses were abolished with simultaneous treatment with NE (5  $\mu$ M). In this study, a prior exposure to NE did not affect the GH responses to subsequent stimulation with GnRH and DA, respectively.

### Discussion and Conclusion

In this study, we have shown that NE inhibits GH release in the goldfish by acting directly at the pituitary cell level. Besides inhibiting basal GH secretion, NE is also capable of blocking the GH-releasing actions of GnRH and DA, which are the known physiological GH-releasing factors in the goldfish. These results suggest that NE, besides being a neurotransmitter, also functions as a GH-release inhibiting factor in the goldfish.

### Acknowledgements

This study was supported by NSERC and CRGC grants.

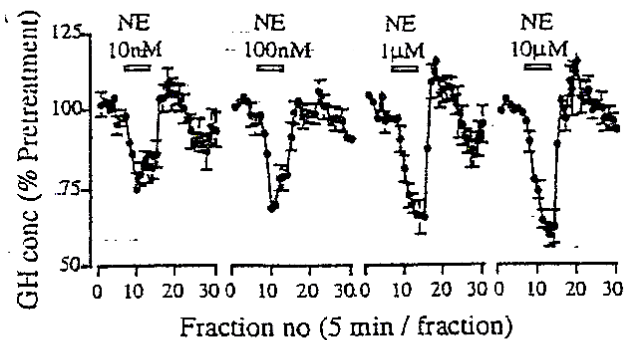


Fig. 1 Effects of norepinephrine (NE) on basal GH release from perfused goldfish cells. Increasing doses (10nM - 10 $\mu$ M) of NE were applied continuously for 30 min as indicated by the white bars. GH data presented (mean  $\pm$  SEM) for each dose of NE are pooled results from four separate perfusion columns (n = 4).

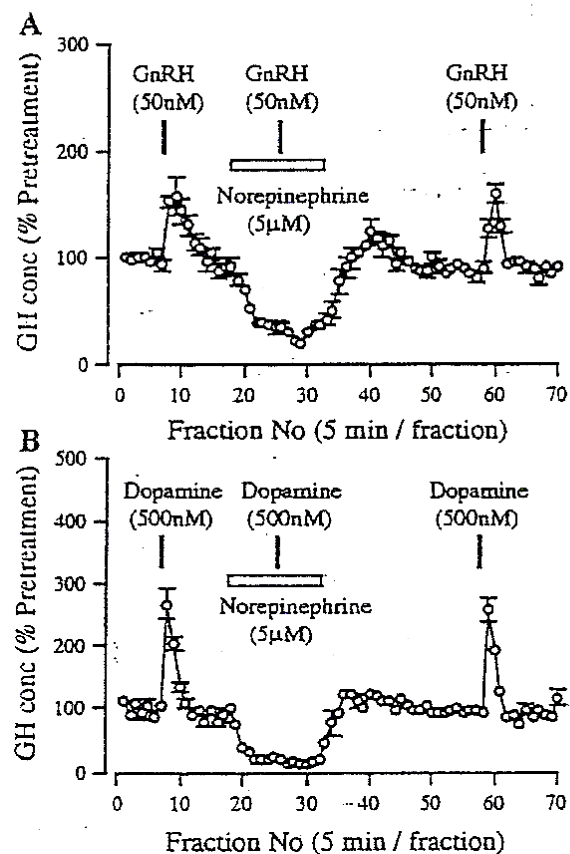


Fig. 2 Effects of norepinephrine (NE) on the GH-releasing actions of GnRH (A) and dopamine (B) in perfused goldfish pituitary fragments. Three consecutive 5-min pulses (black bars) of salmon GnRH (50 nM) and dopamine (500 nM) were applied before, during, and after a 90-min continuous perfusion of NE (5  $\mu$ M, white bars). GH data presented (mean  $\pm$  SEM) are pooled results from four separate columns (n = 4).

### References

- (1) Peter et al. (1990) *J Exp Zool* 4 (suppl):84-89
- (2) Hornby et al. (1990) *Brain Behav Evol* 35:49-64
- (3) Chang et al. (1990) *Gen Comp Endocrinol* 77:256-273
- (4) Marchant et al. (1989) *Gen Comp Endocrinol* 73:458-468