

REFERENCES

1. Melberg, T., R. Burman, and K. Dickstein, *The impact of the 2007 ESC-ACC-AHA-WHF Universal definition on the incidence and classification of acute myocardial infarction: A retrospective cohort study*. Int J Cardiol, 2008.
2. Sucher, R., et al., *Intracellular signaling pathways control mitochondrial events associated with the development of ischemia/ reperfusion-associated damage*. Transpl Int, 2009. **22**(9): p. 922-30.
3. Correa, F., et al., *Relationship between oxidative stress and mitochondrial function in the post-conditioned heart*. J Bioenerg Biomembr, 2008. **40**(6): p. 599-606.
4. Chen, Q., et al., *Ischemic defects in the electron transport chain increase the production of reactive oxygen species from isolated rat heart mitochondria*. Am J Physiol Cell Physiol, 2008. **294**(2): p. C460-6.
5. Farber, J.L., K.R. Chien, and S. Mittnacht, Jr., *Myocardial ischemia: the pathogenesis of irreversible cell injury in ischemia*. Am J Pathol, 1981. **102**(2): p. 271-81.
6. Niizuma, K., H. Endo, and P.H. Chan, *Oxidative stress and mitochondrial dysfunction as determinants of ischemic neuronal death and survival*. J Neurochem, 2009. **109** Suppl 1: p. 133-8.
7. Aon, M.A., et al., *Synchronized whole cell oscillations in mitochondrial metabolism triggered by a local release of reactive oxygen species in cardiac myocytes*. J Biol Chem, 2003. **278**(45): p. 44735-44.

8. Lemasters, J.J., et al., *The mitochondrial permeability transition in cell death: a common mechanism in necrosis, apoptosis and autophagy*. *Biochim Biophys Acta*, 1998. **1366**(1-2): p. 177-96.
9. Hirsch, T., et al., *The apoptosis-necrosis paradox. Apoptogenic proteases activated after mitochondrial permeability transition determine the mode of cell death*. *Oncogene*, 1997. **15**(13): p. 1573-81.
10. Susin, S.A., et al., *The central executioner of apoptosis: multiple connections between protease activation and mitochondria in Fas/APO-1/CD95- and ceramide-induced apoptosis*. *J Exp Med*, 1997. **186**(1): p. 25-37.
11. Nulton-Persson, A.C. and L.I. Szweda, *Modulation of mitochondrial function by hydrogen peroxide*. *J Biol Chem*, 2001. **276**(26): p. 23357-61.
12. Zoratti, M. and I. Szabo, *The mitochondrial permeability transition*. *Biochim Biophys Acta*, 1995. **1241**(2): p. 139-76.
13. Petronilli, V., et al., *The mitochondrial permeability transition, release of cytochrome c and cell death. Correlation with the duration of pore openings in situ*. *J Biol Chem*, 2001. **276**(15): p. 12030-4.
14. Beutner, G., et al., *Complexes between kinases, mitochondrial porin and adenylate translocator in rat brain resemble the permeability transition pore*. *FEBS Lett*, 1996. **396**(2-3): p. 189-95.
15. McEnery, M.W., et al., *Isolation of the mitochondrial benzodiazepine receptor: association with the voltage-dependent anion channel and the adenine nucleotide carrier*. *Proc Natl Acad Sci U S A*, 1992. **89**(8): p. 3170-4.

16. Kinnally, K.W., et al., *Mitochondrial benzodiazepine receptor linked to inner membrane ion channels by nanomolar actions of ligands*. Proc Natl Acad Sci U S A, 1993. **90**(4): p. 1374-8.
17. Wojakowski, W., et al., *Mobilization of CD34/CXCR4+, CD34/CD117+, c-met+ stem cells, and mononuclear cells expressing early cardiac, muscle, and endothelial markers into peripheral blood in patients with acute myocardial infarction*. Circulation, 2004. **110**(20): p. 3213-20.
18. von Vietinghoff, S. and K. Ley, *Homeostatic regulation of blood neutrophil counts*. J Immunol, 2008. **181**(8): p. 5183-8.
19. Cheng, Z., et al., *Granulocyte colony-stimulating factor exacerbates cardiac fibrosis after myocardial infarction in a rat model of permanent occlusion*. Cardiovasc Res, 2008. **80**(3): p. 425-34.
20. Brunner, S., et al., *G-CSF treatment after myocardial infarction: impact on bone marrow-derived vs cardiac progenitor cells*. Exp Hematol, 2008. **36**(6): p. 695-702.
21. Okada, H., et al., *Effect of a long-term treatment with a low-dose granulocyte colony-stimulating factor on post-infarction process in the heart*. J Cell Mol Med, 2008. **12**(4): p. 1272-83.
22. Ostadal, B., et al., *Gender Differences in Cardiac Ischemic Injury and Protection - Experimental Aspects*. Exp Biol Med (Maywood), 2009.
23. van der Laan, A.M., J.J. Piek, and N. van Royen, *Targeting angiogenesis to restore the microcirculation after reperfused MI*. Nat Rev Cardiol, 2009.

24. Kalinkovich, A., et al., *Blood-forming stem cells are nervous: Direct and indirect regulation of immature human CD34(+) cells by the nervous system.* Brain Behav Immun, 2009.
25. Shimoda, K., et al., *Identification of a functional receptor for granulocyte colony-stimulating factor on platelets.* J Clin Invest, 1993. **91**(4): p. 1310-3.
26. Bussolino, F., et al., *Granulocyte- and granulocyte-macrophage-colony stimulating factors induce human endothelial cells to migrate and proliferate.* Nature, 1989. **337**(6206): p. 471-3.
27. Kawada, H., et al., *Nonhematopoietic mesenchymal stem cells can be mobilized and differentiate into cardiomyocytes after myocardial infarction.* Blood, 2004. **104**(12): p. 3581-7.
28. Hasegawa, H., et al., *Cardioprotective effects of granulocyte colony-stimulating factor in swine with chronic myocardial ischemia.* J Am Coll Cardiol, 2006. **47**(4): p. 842-9.
29. Orlic, D., et al., *Mobilized bone marrow cells repair the infarcted heart, improving function and survival.* Proc Natl Acad Sci U S A, 2001. **98**(18): p. 10344-9.
30. Souza, L.M., et al., *Recombinant human granulocyte colony-stimulating factor: effects on normal and leukemic myeloid cells.* Science, 1986. **232**(4746): p. 61-5.
31. Baldo, M.P., et al., *Granulocyte colony-stimulating factor reduces mortality by suppressing ventricular arrhythmias in acute phase of myocardial infarction in rats.* J Cardiovasc Pharmacol, 2008. **52**(4): p. 375-80.

32. Walter, M.R., et al., *Three-dimensional structure of recombinant human granulocyte-macrophage colony-stimulating factor*. J Mol Biol, 1992. **224**(4): p. 1075-85.
33. Abdel-Meguid, S.S., et al., *Three-dimensional structure of a genetically engineered variant of porcine growth hormone*. Proc Natl Acad Sci U S A, 1987. **84**(18): p. 6434-7.
34. Fukunaga, R., et al., *Expression cloning of a receptor for murine granulocyte colony-stimulating factor*. Cell, 1990. **61**(2): p. 341-50.
35. Tamada, T., et al., *Homodimeric cross-over structure of the human granulocyte colony-stimulating factor (GCSF) receptor signaling complex*. Proc Natl Acad Sci U S A, 2006. **103**(9): p. 3135-40.
36. Haniu, M., et al., *Extracellular domain of granulocyte-colony stimulating factor receptor. Interaction with its ligand and identification of a domain in close proximity of ligand-binding region*. Arch Biochem Biophys, 1995. **324**(2): p. 344-56.
37. Dai, Y., et al., *Mobilized bone marrow progenitor cells serve as donors of cytoprotective genes for cardiac repair*. J Mol Cell Cardiol, 2008. **44**(3): p. 607-17.
38. Thomas, J., F. Liu, and D.C. Link, *Mechanisms of mobilization of hematopoietic progenitors with granulocyte colony-stimulating factor*. Curr Opin Hematol, 2002. **9**(3): p. 183-9.
39. Abbott, J.D., et al., *Stromal cell-derived factor-1alpha plays a critical role in stem cell recruitment to the heart after myocardial infarction but is not*

- sufficient to induce homing in the absence of injury.* Circulation, 2004. **110**(21): p. 3300-5.
40. Vandervelde, S., et al., *Signaling factors in stem cell-mediated repair of infarcted myocardium.* J Mol Cell Cardiol, 2005. **39**(2): p. 363-76.
41. Misao, Y., et al., *Importance of recruitment of bone marrow-derived CXCR4+ cells in post-infarct cardiac repair mediated by G-CSF.* Cardiovasc Res, 2006. **71**(3): p. 455-65.
42. Misao, Y., et al., *Modification of post-myocardial infarction granulocyte-colony stimulating factor therapy with myelo-suppressives.* Circ J, 2007. **71**(4): p. 580-90.
43. Leone, A.M., et al., *Endogenous G-CSF and CD34+ cell mobilization after acute myocardial infarction.* Int J Cardiol, 2006. **111**(2): p. 202-8.
44. Fukuhara, S., et al., *G-CSF promotes bone marrow cells to migrate into infarcted mice heart, and differentiate into cardiomyocytes.* Cell Transplant, 2004. **13**(7-8): p. 741-8.
45. Beltrami, A.P., et al., *Evidence that human cardiac myocytes divide after myocardial infarction.* N Engl J Med, 2001. **344**(23): p. 1750-7.
46. Kuwabara, M., et al., *Granulocyte colony-stimulating factor activates Wnt signal to sustain gap junction function through recruitment of beta-catenin and cadherin.* FEBS Lett, 2007. **581**(25): p. 4821-30.
47. Min, J.Y., et al., *Transplantation of embryonic stem cells improves cardiac function in postinfarcted rats.* J Appl Physiol, 2002. **92**(1): p. 288-96.

48. Payne, T.R., et al., *A relationship between vascular endothelial growth factor, angiogenesis, and cardiac repair after muscle stem cell transplantation into ischemic hearts*. J Am Coll Cardiol, 2007. **50**(17): p. 1677-84.
49. Cho, S.W., et al., *Granulocyte colony-stimulating factor treatment enhances the efficacy of cellular cardiomyoplasty with transplantation of embryonic stem cell-derived cardiomyocytes in infarcted myocardium*. Biochem Biophys Res Commun, 2006. **340**(2): p. 573-82.
50. Harada, M., et al., *G-CSF prevents cardiac remodeling after myocardial infarction by activating the Jak-Stat pathway in cardiomyocytes*. Nat Med, 2005. **11**(3): p. 305-11.
51. Okada, H., et al., *Combined therapy with cardioprotective cytokine administration and antiapoptotic gene transfer in postinfarction heart failure*. Am J Physiol Heart Circ Physiol, 2009. **296**(3): p. H616-26.
52. Li, H., et al., *Myocardial survival signaling in response to stem cell transplantation*. J Am Coll Surg, 2009. **208**(4): p. 607-13.
53. Minatoguchi, S., et al., *Acceleration of the healing process and myocardial regeneration may be important as a mechanism of improvement of cardiac function and remodeling by postinfarction granulocyte colony-stimulating factor treatment*. Circulation, 2004. **109**(21): p. 2572-80.
54. Sugano, Y., et al., *Granulocyte colony-stimulating factor attenuates early ventricular expansion after experimental myocardial infarction*. Cardiovasc Res, 2005. **65**(2): p. 446-56.

55. Deten, A., et al., *Changes in extracellular matrix and in transforming growth factor beta isoforms after coronary artery ligation in rats*. J Mol Cell Cardiol, 2001. **33**(6): p. 1191-207.
56. Takemura, G., et al., *Role of apoptosis in the disappearance of infiltrated and proliferated interstitial cells after myocardial infarction*. Circ Res, 1998. **82**(11): p. 1130-8.
57. Creemers, E.E., et al., *Matrix metalloproteinase inhibition after myocardial infarction: a new approach to prevent heart failure?* Circ Res, 2001. **89**(3): p. 201-10.
58. Spinale, F.G., *Matrix metalloproteinases: regulation and dysregulation in the failing heart*. Circ Res, 2002. **90**(5): p. 520-30.
59. Taniyama, Y., et al., *Potential contribution of a novel antifibrotic factor, hepatocyte growth factor, to prevention of myocardial fibrosis by angiotensin II blockade in cardiomyopathic hamsters*. Circulation, 2000. **102**(2): p. 246-52.
60. Lehrke, S., et al., *Aging impairs the beneficial effect of granulocyte colony-stimulating factor and stem cell factor on post-myocardial infarction remodeling*. Circ Res, 2006. **99**(5): p. 553-60.
61. Xiao, J., et al., *4'-Chlorodiazepam, a translocator protein (18 kDa) antagonist, improves cardiac functional recovery during postischemia reperfusion in rats*. Exp Biol Med (Maywood), 2010. **235**(4): p. 478-86.

62. Brennan, J.P., et al., *Mitochondrial uncoupling, with low concentration FCCP, induces ROS-dependent cardioprotection independent of KATP channel activation*. *Cardiovasc Res*, 2006. **72**(2): p. 313-21.
63. Sucher, R., et al., *Intracellular signaling pathways control mitochondrial events associated with the development of ischemia/ reperfusion-associated damage*. *Transpl Int*, 2009.
64. Fridovich, I., *Superoxide anion radical (O₂⁻), superoxide dismutases, and related matters*. *J Biol Chem*, 1997. **272**(30): p. 18515-7.
65. Chance, B., H. Sies, and A. Boveris, *Hydroperoxide metabolism in mammalian organs*. *Physiol Rev*, 1979. **59**(3): p. 527-605.
66. Singal, P.K., et al., *The role of oxidative stress in the genesis of heart disease*. *Cardiovasc Res*, 1998. **40**(3): p. 426-32.
67. Zhu, Q.S., et al., *G-CSF induced reactive oxygen species involves Lyn-PI3-kinase-Akt and contributes to myeloid cell growth*. *Blood*, 2006. **107**(5): p. 1847-56.
68. Olas, B., B. Wachowicz, and A. Buczynski, *The effects of granulocyte colony stimulating factor on chemiluminescence and lipid peroxidation of blood platelets treated with cisplatin*. *Anticancer Drugs*, 2000. **11**(2): p. 79-84.
69. Li, L., et al., *Granulocyte colony-stimulating factor improves left ventricular function of doxorubicin-induced cardiomyopathy*. *Lab Invest*, 2007. **87**(5): p. 440-55.

70. Ueda, K., et al., *Granulocyte colony stimulating factor directly inhibits myocardial ischemia-reperfusion injury through Akt-endothelial NO synthase pathway*. *Arterioscler Thromb Vasc Biol*, 2006. **26**(6): p. e108-13.
71. Leone, A.M., et al., *Mobilization of bone marrow-derived stem cells after myocardial infarction and left ventricular function*. *Eur Heart J*, 2005. **26**(12): p. 1196-204.
72. Ince, H., et al., *Prevention of left ventricular remodeling with granulocyte colony-stimulating factor after acute myocardial infarction: final 1-year results of the Front-Integrated Revascularization and Stem Cell Liberation in Evolving Acute Myocardial Infarction by Granulocyte Colony-Stimulating Factor (FIRSTLINE-AMI) Trial*. *Circulation*, 2005. **112**(9 Suppl): p. I73-80.
73. Kuethé, F., et al., *Treatment with granulocyte colony-stimulating factor for mobilization of bone marrow cells in patients with acute myocardial infarction*. *Am Heart J*, 2005. **150**(1): p. 115.
74. Kang, H.J., et al., *Differential effect of intracoronary infusion of mobilized peripheral blood stem cells by granulocyte colony-stimulating factor on left ventricular function and remodeling in patients with acute myocardial infarction versus old myocardial infarction: the MAGIC Cell-3-DES randomized, controlled trial*. *Circulation*, 2006. **114**(1 Suppl): p. I145-51.
75. Kang, H.J., et al., *Effects of intracoronary infusion of peripheral blood stem-cells mobilised with granulocyte-colony stimulating factor on left ventricular systolic function and restenosis after coronary stenting in myocardial*



- infarction: the MAGIC cell randomised clinical trial*. Lancet, 2004. **363**(9411): p. 751-6.
76. Wang, Y., et al., *Changes in circulating mesenchymal stem cells, stem cell homing factor, and vascular growth factors in patients with acute ST elevation myocardial infarction treated with primary percutaneous coronary intervention*. Heart, 2006. **92**(6): p. 768-74.
77. Ohtsuka, M., et al., *Cytokine therapy prevents left ventricular remodeling and dysfunction after myocardial infarction through neovascularization*. FASEB J, 2004. **18**(7): p. 851-3.
78. Mitchell, P., *Chemiosmotic coupling in oxidative and photosynthetic phosphorylation*. Biol Rev Camb Philos Soc, 1966. **41**(3): p. 445-502.
79. Liu, X., et al., *Induction of apoptotic program in cell-free extracts: requirement for dATP and cytochrome c*. Cell, 1996. **86**(1): p. 147-57.
80. Kroemer, G., N. Zamzami, and S.A. Susin, *Mitochondrial control of apoptosis*. Immunol Today, 1997. **18**(1): p. 44-51.
81. Hiraumi, Y., et al., *Granulocyte colony-stimulating factor protects cardiac mitochondria in the early phase of cardiac injury*. Am J Physiol Heart Circ Physiol, 2009. **296**(3): p. H823-32.
82. Larche, J., et al., *Inhibition of mitochondrial permeability transition prevents sepsis-induced myocardial dysfunction and mortality*. J Am Coll Cardiol, 2006. **48**(2): p. 377-85.
83. Walker, J.M., *The bicinchoninic acid (BCA) assay for protein quantitation*. Methods Mol Biol, 1994. **32**: p. 5-8.

84. Chelli, B., et al., *Peripheral-type benzodiazepine receptor ligands: mitochondrial permeability transition induction in rat cardiac tissue*. *Biochem Pharmacol*, 2001. **61**(6): p. 695-705.
85. Ruiz-Meana, M., et al., *Mitochondrial Ca²⁺ uptake during simulated ischemia does not affect permeability transition pore opening upon simulated reperfusion*. *Cardiovasc Res*, 2006. **71**(4): p. 715-24.
86. Novalija, E., et al., *Anesthetic preconditioning improves adenosine triphosphate synthesis and reduces reactive oxygen species formation in mitochondria after ischemia by a redox dependent mechanism*. *Anesthesiology*, 2003. **98**(5): p. 1155-63.
87. Tong, V., et al., *Valproic acid II: effects on oxidative stress, mitochondrial membrane potential, and cytotoxicity in glutathione-depleted rat hepatocytes*. *Toxicol Sci*, 2005. **86**(2): p. 436-43.
88. Di Lisa, F., et al., *Mitochondrial membrane potential in single living adult rat cardiac myocytes exposed to anoxia or metabolic inhibition*. *J Physiol*, 1995. **486** (Pt 1): p. 1-13.
89. Tompkins, A.J., et al., *Mitochondrial dysfunction in cardiac ischemia-reperfusion injury: ROS from complex I, without inhibition*. *Biochim Biophys Acta*, 2006. **1762**(2): p. 223-31.
90. Zorov, D.B., M. Juhaszova, and S.J. Sollott, *Mitochondrial ROS-induced ROS release: an update and review*. *Biochim Biophys Acta*, 2006. **1757**(5-6): p. 509-17.

91. Matejikova, J., et al., *Mitochondrial KATP opening confers protection against lethal myocardial injury and ischaemia-induced arrhythmias in the rat heart via PI3K/Akt-dependent and -independent mechanisms*. Can J Physiol Pharmacol, 2009. **87**(12): p. 1055-62.
92. Moncada, S., *Mitochondria as pharmacological targets*. Br J Pharmacol, 2010. **160**(2): p. 217-9.
93. Hausenloy, D.J., M.R. Duchon, and D.M. Yellon, *Inhibiting mitochondrial permeability transition pore opening at reperfusion protects against ischaemia-reperfusion injury*. Cardiovasc Res, 2003. **60**(3): p. 617-25.
94. Azzone, G.F. and A. Azzi, *Volume changes in liver mitochondria*. Proc Natl Acad Sci U S A, 1965. **53**(5): p. 1084-9.
95. Crompton, M., *The mitochondrial permeability transition pore and its role in cell death*. Biochem J, 1999. **341** (Pt 2): p. 233-49.
96. Brady, N.R., et al., *A wave of reactive oxygen species (ROS)-induced ROS release in a sea of excitable mitochondria*. Antioxid Redox Signal, 2006. **8**(9-10): p. 1651-65.
97. Vanden Hoek, T.L., et al., *Reactive oxygen species released from mitochondria during brief hypoxia induce preconditioning in cardiomyocytes*. J Biol Chem, 1998. **273**(29): p. 18092-8.
98. Zorov, D.B., et al., *Reactive oxygen species (ROS)-induced ROS release: a new phenomenon accompanying induction of the mitochondrial permeability transition in cardiac myocytes*. J Exp Med, 2000. **192**(7): p. 1001-14.

99. Cortassa, S., et al., *A mitochondrial oscillator dependent on reactive oxygen species*. Biophys J, 2004. **87**(3): p. 2060-73.
100. Carrao, A.C., et al., *Stimulation of coronary collateral growth by granulocyte stimulating factor: role of reactive oxygen species*. Arterioscler Thromb Vasc Biol, 2009. **29**(11): p. 1817-22.
101. Kroemer, G., L. Galluzzi, and C. Brenner, *Mitochondrial membrane permeabilization in cell death*. Physiol Rev, 2007. **87**(1): p. 99-163.

APPENDICES

APPENDICES A

List of chemicals and materials used in the study

Chemicals	Sources
Granulocyte colony stimulating factor (G-CSF) Ltd	Roche Thailand
Sucrose	Sigma
TES	Gibthai
EGTA	Sigma
Sodium bicinchoninate	Sigma
$\text{Na}_2\text{CO}_3 \cdot \text{H}_2\text{O}$	Sigma
Sodium tartrate (dihydrate)	Sigma
NaOH	Merck
NaHCO_3	Sigma
$\text{CuSO}_4 \cdot 5\text{H}_2\text{O}$	Sigma
Potassium chloride (KCl)	Sigma

HEPES	Sigma
KH ₂ PO ₄	Sigma
The dye 5,5',6,6'-tetrachloro-1,1',3,3'-	Sigma
Tetraethylbenzimidazolcarbocyanine iodide (JC-1)	Sigma
Dichlorohydro-fluorescein diacetate (DCFDA)	Sigma
Dimethyl sulfoxide (DMSO)	Sigma
CsA	Sigma
4'-Cl-DZP	Sigma

APPENDICES B

List of instruments used in the study

Instruments	Company
Automatic pipette	Gibthai
Balance	Mettler-Toledo
Carbon dioxide incubator	SHEL LAB
Centrifuge	Sigma
Deionized-water manufacturing machine promotion	Scientific
Light microscope	Gibthai
Transmission electron microscope	Gibthai
Fluorescent microplate reader	Biotex
Pasteur pipette	Pyrex
PH meter	Gibthai
Spectrofluorometer	Shimadzu
Sterile tube (15 and 50 ml)	NEPTUNE
Vortex mixture	Wisemix

Water bath

Gibthai

Homogenizer

BECTHAI

96-well plate

Nunclon

APPENDIX C

Preparation of some chemicals and buffers

1. G-CSF

Trade name: Neupogen

Other names: G-CSF, granulocyte-colony stimulating factor

G-CSF is used to stimulate the production of granulocytes (a type of white blood cell) in patients undergoing therapy that will cause low white blood cell counts. This medication is used to prevent infection and neutropenic (low white blood cells) fevers caused by chemotherapy.

How G-CSF is given:

- G-CSF may be given by subcutaneous (the layer between the skin and muscle) injection or infused into a vein (intravenous, IV).

- G-CSF is generally given on a daily basis. The number of days you receive filgrastim will be prescribed by your doctor.

Side effect:

1. Most people do not experience all of the side effects listed.
2. Side effects are often predictable in terms of their onset and duration.
3. Side effects are almost always reversible and will go away after treatment is complete.

4. There are many options to help minimize or prevent side effects.
5. There is no relationship between the presence or severity of side effects and the effectiveness of the medication.

2. DCFDA

Reactive oxygen species (ROS) were detected with the dichlorohydrofluorescein diacetate (DCFDA). DCFDA, a redox-sensitive fluorescent probe, was purchased from (Sigma). DCFDA passes through cell membranes where it is cleaved by esterases to DCF and becomes activated by oxidation. Fluorescence of the samples was measured using a fluorescent microplate reader.

3. Dye 5,5',6,6'-tetrachloro-1,1',3,3'-tetraethylbenzimidazolcarbocyanine iodide (JC-1)

JC-1, a redox-sensitive fluorescent probe, was purchased from (Sigma) for study protocol. JC-1 was dissolved in dimethyl sulphoxide (DMSO), fractionated in small aliquots and stored at -20°C. This fluorescent dye was equipped with two separate photomultipliers for collecting simultaneously fluorescence emitted at two different wavelengths during study.

4. Isolated buffer

Isolated buffer contains sucrose 300 mM, TES 5 mM and EGTA 0.2 mM (pH 7.2) (4°C).

5. Respiration buffer

Respiration buffer (containing 100 mM KCl, 50 mM sucrose, 10 mM HEPES, and 5 mM KH_2PO_4 (pH 7.4) (37°C).



CURRICURUM VITAE

Name Miss Savitree Thummasorn

Date of Birth January 10, 1984

Education

- March, 1998 Certificate of junior high school, Phadungpanya school, Tak, Thailand
- March, 2001 Certificate of junior high school, Phadungpanya school, Tak, Thailand
- March, 2006 Bachelor of Science, Faculty of Associate medical science, Chiang Mai university, Thailand

Presentation at National and International Meeting

May, 2010 Oral presentation in the 39th Physiological society of Thailand's Annual Conference 2010, Pattaya, Chonburi, Thailand (Received Oral Presentation Award)

