



KEMENTERIAN PENDIDIKAN DAN KEBUDAYAAN  
UNIVERSITAS AIRLANGGA  
FAKULTAS KEDOKTERAN

Kampus A Jalan Mayjen Prof.Dr.Moestopo 47 Surabaya 60131 Telepon (031) 5030252-3 Faks. 5022472  
Website : <http://www.fk.unair.ac.id> e-mail : [dekan@fk.unair.ac.id](mailto:dekan@fk.unair.ac.id)

**SURAT KETERANGAN DEKAN**  
**No. 0132/UN3.1.1/KP/2020**

Yang bertandatangan di bawah ini :

Nama : Prof. Dr. Soetojo, dr, Sp.U(K)  
NIP : 195606081986121001  
Pangkat/Golongan Ruang : Pembina Utama Madya (Gol. IV/d)  
Jabatan : Dekan/Guru Besar  
Unit Organisasi : Fakultas Kedokteran Unair

Menerangkan bahwa:

Nama : Viskasari Pintoko Kalanjati, dr., M.Kes., Ph.D.  
NIP : 197603202005012003  
Pangkat/Gol Ruang : Pembina (Gol. IV/a)  
Jabatan Fungsional : Lektor Kepala/ 1 Agustus 2017  
Unit Kerja : Departemen Anatomi dan Histologi Fakultas Kedokteran Unair

Telah melaksanakan kegiatan pendidikan dan pengajaran pada mahasiswa Fakultas Kedokteran Universitas Airlangga sebagai **Penguji Tugas Akhir**. (Daftar Terlampir).

Demikian surat keterangan ini dibuat untuk dapat dipergunakan sebagaimana mestinya.



Surabaya  
Dekan  
Fakultas Kedokteran Unair  
Prof. Dr. Soetojo, dr, Sp.U(K)  
NIP. 195606081986121001

**UNIVERSITAS AIRLANGGA**  
**FAKULTAS KEDOKTERAN**

LAMPIRAN :

Surat Keterangan Dekan Fakultas Kedokteran Universitas Airlangga No : 0132/UN3.1.1/KP/2020 Tanggal 3 Februari 2020 Tentang Staf Pengajar Departemen Anatomi dan Histologi yang diberi tugas melaksanakan kegiatan Pendidikan dan Pengajaran Tugas Sebagai Penguji

No	STAF PENGAJAR	KODE SUB UNSUR	UJIAN	TAHUN	SKS
1	Viskasari Pintoko Kalanjati, dr., M.Kes., Ph.D. NIP: 197603202005012003 Pembina (Gol. IV/a)	A.1.	Bertugas Menguji Ujian Terbuka Mahasiswa S3 FK Unair: 1. Santika Rentika Hadi, Drs., M.Kes 2. Arif Rachman, drg., MM., MT., Sp.Pros 3. M. Fathul Qorib, dr., SpKFR		0,5 0,5 0,5
<b>Jumlah</b>					<b>1,5</b>



Dekan  
Fakultas Kedokteran Universitas Airlangga  
Prof. Dr. Soetoyo, dr., Sp.U(K) *[Signature]*  
NIP. 19560608 198612 1 001

**BUKU DISERTASI RINGKAS**

**PENGARUH *DETRAINING* TERHADAP POLA  
*EKSPRESI HSP60*, DAN *HSP70* PADA SEL OTOT  
JANTUNG *RATTUS NORVEGICUS WISTAR***



**SANTIKA RENTIKA HADI**

**PROGRAM STUDI ILMU KEDOKTERAN JENJANG DOKTOR  
FAKULTAS KEDOKTERAN UNIVERSITAS AIRLANGGA  
SURABAYA  
2018**

**PENGARUH *DETRAINING* TERHADAP POLA  
EKSPRESI *HSP60*, DAN *HSP70* PADA SEL OTOT  
JANTUNG *RATTUS NORVEGICUS WISTAR***

**RINGKASAN DISERTASI**

Untuk memperoleh Gelar Doktor  
Dalam Program Studi Ilmu Kedokteran Jenjang Doktor  
Pada Fakultas Kedokteran Universitas Airlangga dan  
Dipertahankan di hadapan Panitia Ujian Akhir  
Tahap 2 (Terbuka)

Hari : Kamis  
Tanggal : 22 Pebruari 2018  
Pukul : 10.00 Wib.

*Oleh :*

**Santika Rentika Hadi  
091070104**

**PROGRAM STUDI ILMU KEDOKTERAN JENJANG DOKTOR  
FAKULTAS KEDOKTERAN UNIVERSITAS AIRLANGGA  
SURABAYA  
2018**

## ABSTRACT

### THE EFFECT OF DETRAINING TOWARDS THE PATTERN EXPRESSION OF HSP60 AND HSP70 TO THE CARDIAC MUSCULAR CELL OF *RATTUS NORVEGICUS WISTAR*

Santika Rentika Hadi

**Objective:** The goals of this research was to find out the decline pattern of HSP60 and HSP70 in cardiac muscular as the effect of one, two, three and four weeks period of detraining and the interconnection to the MDA (ROS) expression.

**Material and Methods:** *Rattus norvegicus wistar* trained for endurance swimming (aerobic) for six weeks so that they became trained *rattus norvegicus wistar*, continued by giving treatment of detraining based on the time allocation.

**Results:** The conclusion of this research were as follows: (1) Expression HSP60 to rattus' cardiac muscular decrease after detraining in four weeks. (2) Expression HSP70 to rattus' cardiac muscular decrease after detraining for in three weeks and keep in low level in four weeks detraining. (3) Expression of MDA in rattus' cardiac muscular decrease in one week detraining and is kept in low level in four weeks detraining. (4) Expression of MDA and HSP60 in rattus' cardiac muscular decrease after doing physical exercises in one week and is kept in low level in ten weeks, but the expression of HSP70 is kept high. It is suggested that (1) Physical exercise with the intensity of 60-80% from maximum working capacity should be continuously well maintained. (2) Detraining period pause should not be more than two weeks. (3) Further research is needed for conforming the apoptosis of cardiac muscular cell which could happen because of detraining.

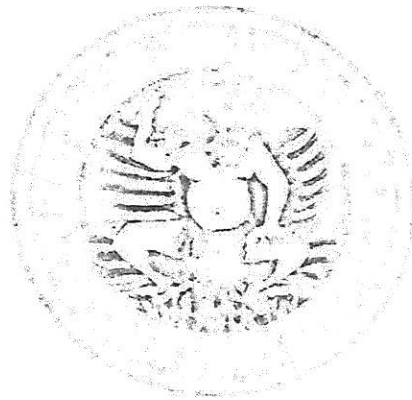
**Conclusion:** Detraining lowers the MDA (stressor) which also decreases Hsp60 and Hsp70 (protector) consequently the heart is easily disturbed when recovering physical activity after the detraining period, it is recommended to restart physical exercise with a light load.

**Key words:** *detraining*, HSP60, HSP70, MDA

**BUKU DISERTASI RINGKAS**

**MEKANISME PERCEPATAN REGENERASI TULANG  
MANDIBULA PADA IMPLANTASI YTTRIA-  
TETRAGONAL ZIRCONIA POLYCRYSTAL YANG  
DISEEDING DENGAN HUMAN ADIPOSE  
DERIVED MESENCHYMAL STEM CELL**

**(PENELITIAN EKSPERIMENTAL LABORATORIS PADA TIKUS PUTIH JENIS *WISTAR*)**



**ARIF RACHMAN**

**PROGRAM STUDI ILMU KEDOKTERAN JENJANG DOKTOR  
FAKULTAS KEDOKTERAN UNIVERSITAS AIRLANGGA  
SURABAYA**

**2019**

**MEKANISME PERCEPATAN REGENERASI TULANG  
MANDIBULA PADA IMPLANTASI YTTRIA-  
TETRAGONAL ZIRCONIA POLYCRYSTAL YANG  
DISEEDING DENGAN HUMAN ADIPOSE  
DERIVED MESENCHYMAL STEM CELL**

(PENELITIAN EKSPERIMENTAL LABORATORIS PADA TIKUS PUTIH JENIS *WISTAR*)

**RINGKASAN DISERTASI**  
Untuk memperoleh Gelar Doktor  
dalam Program Studi Ilmu Kedokteran Jenjang Doktor  
pada Fakultas Kedokteran Universitas Airlangga  
dan dipertahankan di hadapan Panitia Ujian Akhir  
Tahap II (Terbuka)

Hari : Senin  
Tanggal : 25 Februari 2019  
Pukul : 10.00

Oleh:

**ARIF RACHMAN**  
011617017301

**PROGRAM STUDI ILMU KEDOKTERAN JENJANG DOKTOR  
FAKULTAS KEDOKTERAN UNIVERSITAS AIRLANGGA  
SURABAYA  
2019**

## ABSTRACT

### MECHANISM OF ACCELERATION OF REGULARATION OF MANDIBULA BONE IN YTTRIA-TETRAGONAL ZIRCONIA IMPLANTATIONS POLYCRYSTAL THAT IS SEEDED WITH HUMAN ADIPOSE DERIVED MESENCHYMAL STEM CELL (EXPERIMENTAL LABORATORY RESEARCH ON WISTAR TYPE WHITE RATS)

**Introduction:** Addition of human Adipose Mesenchymal Stem Cell (hADMSC) to Y-TZP to treat implant failure due to biological factors (peri-implantitis and mucositis) and technical factors; namely cases of immediate implants, age (elderly), hormonal factors (menopause), and immunocompromised factors (diabetes), as well as biomaterials that are not biocompatible. The scaffold is a place for regeneration of new bone and bone tissue growths in tissue engineering applications. hADMSC is a multipotent cell which can differentiate into osteogenic, chondrogenic, and adipogenic. Y-TZP has been shown to have several advantages over other ceramics because of its hard nature, namely fracture toughness and high flexural strength.

**Objective:** This study aimed to explain the mechanism for accelerating mandibular bone regeneration at Y-TZP implantation that seeded with hADMSC which was evaluated qualitatively (SEM) and quantitatively (HE and IHC) and a mixture of qualitative and quantitative (Micro-CT).

**Methods:** This research involved several processes in vitro and in vivo, namely Y-TZPS manufacture process, XRD examination, differentiation and characterization of hADMSC, SEM observation, Toxicity Test, Micro-CT, HE and then IHC.

**Results:** The results of the XRD examination showed that Y-TZPSs had sharp peaks. It suggests that they had high crystal purity. The presence of hADMSC in the Y-TZP scaffold apparently accelerated the osseointegration process of the implant with the mechanism of accelerating mandibular bone regeneration in the Y-TZP-hADMSC scaffold effect at week 1 with the Y-TZP scaffold seeded with hADMSC, increasing



BMP2 which can increase osteoblasts. Increased osteoblasts can increase bone density significantly.

**Conclusions:** Y-TZPS is expected to be used as implantable biomaterials that can be seeded with hADMSCs so that they will have osseointegration potential in the first week.

**Keywords:** Mesenchymal Stem Cell, Y-TZP ceramic, Bone Regeneration, Osseointegration, Tissue Engineering

**BUKU DISERTASI RINGKAS**

**PERAN CDK5 DAN TRPV1 DALAM SIGNAL  
TRANSDUKSI RESISTENSI MELOKSIKAM PADA  
TIKUS PUTIH YANG MENGALAMI NYERI KRONIK**



*Pain  
wind  
up?*

*fehmb  
moro neuro  
surgery*

**MOHAMMAD FATHUL QORIB**

**PROGRAM STUDI ILMU KEDOKTERAN JENJANG DOKTOR**

**FAKULTAS KEDOKTERAN UNIVERSITAS AIRLANGGA**

**SURABAYA**

**2019**

**PERAN CDK5 DAN TRPV1 DALAM SIGNAL  
TRANSDUKSI RESISTENSI MELOKSIKAM PADA  
TIKUS PUTIH YANG MENGALAMI NYERI KRONIK**

**RINGKASAN DISERTASI**

**Untuk memperoleh Gelar Doktor  
Dalam Program Studi Ilmu Kedokteran Jenjang Doktor  
Pada Fakultas Kedokteran Universitas Airlangga  
Dan dipertahankan di hadapan Panitia Penguji Akhir**

**Tahap II (Terbuka)**

**Hari : Selasa  
Tanggal : 17 September 2019**

**MOHAMMAD FATHUL QORIB  
011317017312**

**PROGRAM STUDI ILMU KEDOKTERAN JENJANG  
DOKTOR  
FAKULTAS KEDOKTERAN UNIVERSITAS  
AIRLANGGA  
SURABAYA  
2019**

## ABSTRACT

### The Role of Cdk5 and TRPV1 on Meloxicam Resistance Signal Transduction of Rat Experiencing Chronic Pain

Mohammad Fathul Qorib

**Background.** Chronic pain is a common case and become a serious problem. Chronic pain management are inconclusive. One of the common treatment option is NSAID. Treatment Failed are around 34-79% of total case. These treatment failed suspected as NSAID resistance. Treatment failed can be caused by a some molecule that make NSAID loss of efficacy. Cdk5 is one of the molecule that active in chronic pain condition. Cdk5 can increase transmembrane insertion and activate TRPV1.

**Objective.** The aim of this research is analyzed the role of Cdk5 and TRPV1 in NSAID resistance of chronic pain rat.

**Method.** This research used 42 Wistar rats as a subject and divided into 6 groups with random allocation method and factorial design. Meloxicam treatment was given orally every day for 7 days after rats have a chronic pain (28 days). Chronic pain induction used a CFA injection.

**Results.** Cdk5 and TRPV1 expression at the dorsal root ganglia of chronic pain groups are increase. Cdk5 and TRPV1 expression are highest in the chronic pain with meloxicam (dose D) treatment group. No significant different of pain threshold and inflammation sign between treatment and no treatment groups after chronic pain occurred.

**Conclusion.** Chronic pain can induce Cdk5 and TRPV1 expression, and induced by meloxicam treatment. Cdk5 and TRPV1 have a positive correlation with meloxicam resistance.

**Key Words :** Chronic pain, Cdk5, TRPV1, Meloxicam

resistance