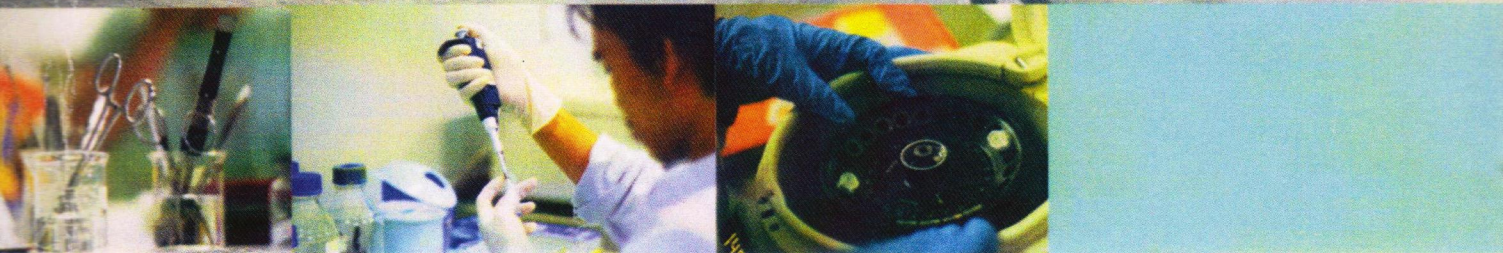
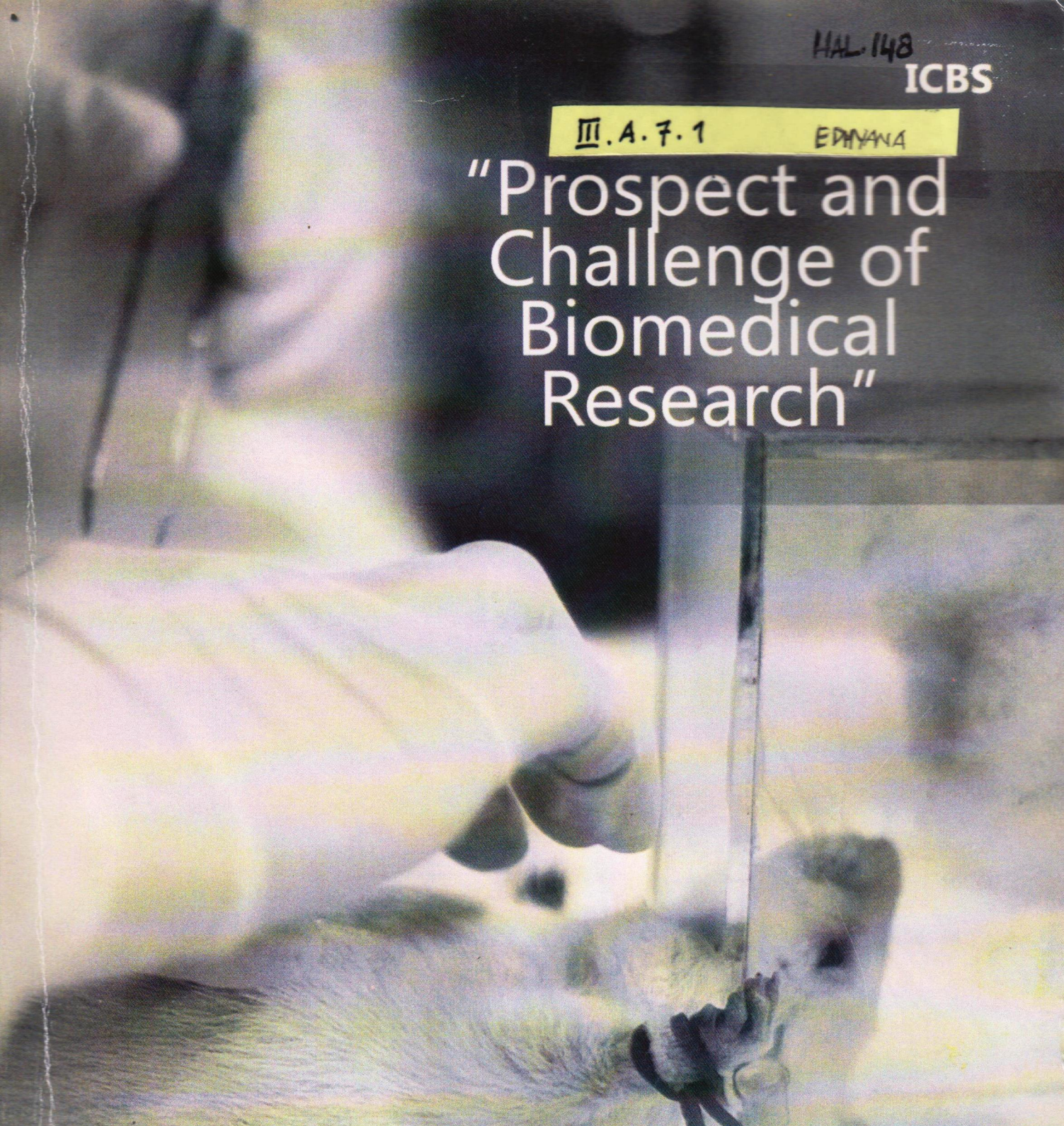


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EDYANA

# "Prospect and Challenge of Biomedical Research"



International  
Conference on  
Biomedical  
Science 2012



**SCHOOL OF LIFE SCIENCES AND TECHNOLOGY (SITH)  
INSTITUT TEKNOLOGI BANDUNG**

**This Certificate is Awarded to**

**Dr. EDHYANA SAHIRATMADJA**

as

**PARTICIPANT  
& POSTER PRESENTER**  
at

**International Conference on Biomedical Sciences :  
Prospect And Challenge of Biomedical Research**

**27<sup>th</sup> – 28<sup>th</sup> February 2012 Bandung, INDONESIA**

Chairperson of ICBS 2012,



Dr. rer. nat. Marselina Irasonia Tan



Prof. Dr. Tati Suryati Syamsudin, MS.DEA



# Human Papilloma Virus Genotype and the *IFNG* Susceptibility



2012  
International Conference on  
Biomedical Science

## among Cervical Cancer Patients in Bandung

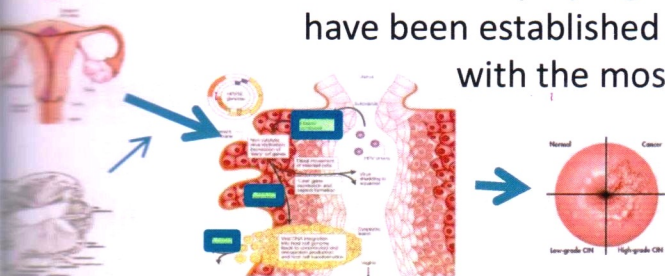
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**Background:** Persistent infections by high-risk types of human papillomavirus (HPV) have been established as the etiological agent of cervical cancer, with the most prevalent types are HPV-16 and HPV-18.



- ✓ There is a strong evidence that the defense mechanism to HPV is a local response.
- ✓ The majority of the women will clear the infection without overt clinical disease.
- ✓ Those who develop lesions mount an effective cell mediated immune response, and hence the lesions will regress.
- ✓ Interferon gamma (IFN- $\gamma$ ) is one of the key regulatory cytokines that influence the HPV clearance.
- ✓ The IFN- $\gamma$  production may be impaired by the *IFNG* gene dysfunction leading to the cervical malignant progression.

Human papillomavirus (HPV) types  
 Alpha 7 Species: HPV 18, 39, 45, 59, 68, 70, c85  
 Alpha 9 Species: HPV 16, 31, 33, 35, 52, 58, 67  
 Alpha 10 Species HPV 6, 11, 13, 44, 55, 74,  
 PcPV, CCPV

**M**  
 analyse distribution of *IFNG* +874T/A polymorphism  
 gene susceptibility to cervical cancer

### METHOD

HPV Genotyping: Linear Array  
*IFNG* polymorphism: ARMS PCR

### RESULT

	NK SCC n=25
HPV-16 (single infection)	3
HPV-16, -18	5
HPV-16, -18, -45	1
HPV-16, ----, -45, -52/33/38/58	6
HPV-16, -18, -45, -52/33/38/58	5
HPV-16, -18, ----, -52/33/38/58	2
HPV-16, ----, -51	1
HPV-16, -18, ----, -51	1
HPV-16, ----, ----, -61*	1

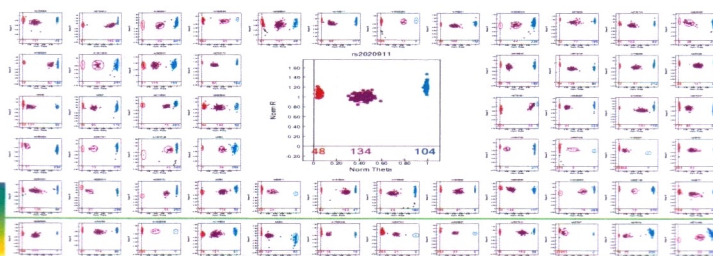
Figure 1: Distribution of HPV infection among  
 Keratinizing Squamous Cell Carcinoma

	allele / genotype	Frequency Cervical cancer n (%)	Frequency NILM n (%)	P
IFN +874 T/A	T	50 (39.1)	28 (33.3)	0.39
	A	78 (60.9)	56 (66.7)	
	TT	9 (14.1)	3 (7.1)	
	TA	32 (50.0)	22 (52.4)	
	AA	23 (35.9)	17 (40.5)	

Table 2: Frequency of *IFNG* +874 T/A in patient with cervical cancer and normal PAP smear

### FUTURE DIRECTION

Polymorphisms of gene susceptible methods  
 → BEADSXPRESS Genotypers (min 48 SNPs)  
 available in Universitas Padjadjaran



**FULL PAPER**  
**ORAL and POSTER PRESENTATION**

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Susceptibility among Cervical Cancer Patients in Bandung**

By

**Edhyana Sahiratmadja<sup>1,2</sup>, Ani Melani Maskoen<sup>2</sup>, Ramdan Panigoro<sup>1</sup>, Herman Susanto<sup>3</sup>**

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**School of Life Sciences and Technology  
Institut Teknologi Bandung  
In Bandung  
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### Abstract

Several persistent infections by high-risk types of human papillomavirus (HPV) have been established as the etiological agent of cervical cancer, with the most prevalent types are HPV-16 and HPV-18. There is a strong evidence that the defense mechanism to HPV is a local response. The majority of the women will clear the infection without overt clinical disease. Those who develop lesions mount an effective cell mediated immune response, and hence the lesions will regress. Interferon gamma (IFN- $\gamma$ ) is one of the key regulatory cytokines that influence the HPV clearance. The IFN- $\gamma$  production may be impaired by the *IFNG* gene dysfunction leading to the cervical malignant progression. The distribution of *IFNG* +874T/A polymorphism in gene susceptibility to cervical cancer was analysed. Interestingly, geographical variation in HPV genotypes might have impacted for designing diagnostic tests and vaccine, therefore, early HPV infection detection may minimize the risk of cancer development. However, it has been taken into account that the tests available might yield a different genotype distribution in the population. Our study showed a different result of two commercial kits. In a poor resource area where HPV genotyping is expensive, a cheaper alternative for HPV genotyping detection might have a beneficial impact.

**Key words:** Cervical cancer, HPV genotypes, *IFNG* +874 T>A