



## Epidermal Growth Factor Receptor (EGFR) as Therapeutic Target in Colorectal Cancer

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Colorectal cancer is the fourth most frequent malignant disease in the world. Estimation of new cases and mortality are 1.023.000 and 529.000 annually. There is an increase of colorectal cancer incidence in Indonesia, but no exact number available.

The problems in management of colorectal cancer are patients came in advanced stage, refractory cytostatics regiment, adverse reaction to cytostatics. Alternative strategy uses an agent that act at specific site; for instance Epidermal Growth Factor Receptor (EGFR) inhibitor.

EGFR have specific ligand including EGF (epidermal growth factor), bFGF (basic fibroblast growth factor), VEGF(vascular endothelial growth factor) and TGF- $\beta$  (transforming growth factor- $\beta$ ). They have important role in growth dan survival of colorectal cancer. EGFR expression in colorectal cancer is associated with aggressive disease and poor prognosis. EGFR stimulates tumor growth and progression through several mechanism i.e. proliferation, angiogenesis, invasion, metastasis, apoptosis inhibition, adhesion and differentiation.

EGFR is specific rational target. Monoclonal antibody (mAbs) directed against EGFR through several mechanism: (1) extracellular binding; (2) internalization of receptor-anti-body complexes; (3) inhibition of EGFR signalling pathways; and (4) potential stimulation of an immunological response.

Tyrosine kinase inhibitors (TKIs) directed against EGFR through several mechanisms: (1) intracellular binding; (2) prevention of tyrosine kinase activation; and (3) inhibition of EGFR signalling pathways.

There are many trials of cetuximab as EGFR inhibitor. Cetuximab is known as IMC-25 or C255, monoclonal antibody chimeric that is specifically directed against EGFR.

The Food and Drug Administration and Swiss Medical Control Agency approved cetuximab for the irinotecan-refractory colorectal cancer. Bevacizumab is humanized antibody that inhibit VEGF action. The Food and Drug Administration approved the use of bevacizumab in combination with any intravenous fluorouracil-containing regimen as initial therapy for patients with advanced colorectal cancer. There is small gefitinib trial for colorectal cancer treatment.

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## Clinical Characteristics of ARMD Patients at Cicendo Eye Hospital Bandung

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Age Related Macular Degeneration (ARMD) affects the central area of the retina (macula). ARMD is the leading cause of severe irreversible central vision loss. The aim of this study is to determine the clinical characteristics of ARMD at Cicendo Eye Hospital.

This prospective descriptive study was conducted on newly diagnosed ARMD on July - November 2005 in Cicendo Eye Hospital. Ninety nine patients (196 eyes) consists of 45,5% males and 54,5% females, ages from

diagnoses were: early - 63 (32,1%) eyes; intermediate - 43 (21,9%) eyes; advanced - 90 (45,9%) eyes. Advanced cases was subdivided into two categories: non neovascular (dry) - 12 (6,1%) eyes and neovascular (wet)- 78 (39,8%) eyes. 24 (26,7%) eyes with cicatrix disciformis complication and 45 (40%) had FFA alone. 34,9% patients with low vision and 27,8% with blindness. A higher rate of neovascular ARMD was noted in Cicendo Eye Hospital.

**Key words:** ARMD, neovascular, visual acuity.

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## Mutagenicity Test of Benzo( $\alpha$ )pyrene by Microneucleus Method on Albino Mice (*Mus musculus*) Bone Marrow

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Benzo ( $\alpha$ ) pyrene (BP) is a carcinogenic compound that is supposed to be able to induce chromosomal damage. The mutagenic effect of BP has been studied using MN test on polychromatic erythrocyte (PCE) cells of albino mice femur bone marrow. Mice were injected with 0.1 ml of 0.3% (b/v) BP subcutaneously at the shoulder, every day at the same time for ten days. After 120 days, treated and non-treated mice were killed by cervical dislocation. Their femur bone marrow cells were prepared on object glass by smear technique followed by Giemsa's staining. The appearance of micronucleus (MN) in PCE cells were examined microscopically by the magnification of 2000. The amount of MN in PCE (MNPCE) were evaluated in 1000 PCE cells and called MNPCE frequency. BP treatment could increase MNPCE frequency up to  $38.82 \pm 8.70$  (n=10) per 1000 PCE cells compared to MNPCE frequency of  $2.19 \pm 0.99$  per 1000 PCE cells in control group (n=10). The significant increase of MNPCE frequency indicated the relatively high mutagenic effect of BP.

**Key words:** benzo ( $\alpha$ ) pyrene, micronucleus, carcinogenic.

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