FOLIA MEDICA INDONESIANA
Vol. 39 No. 2 April - June 2003

Editorial ........................................................................................................................................ 73

Opinion:
Certi OR SHOULD HUMANS BE CLONED? .................................................................................... 74
(Purnomo Suryahudeya)

CORRELATIONS BETWEEN PSYCHOLOGICAL STRESSOR AND SEVERE PREECLAMPSIA
(SPE) ............................................................................................................................................. 76
(JB Dalono)

POST-ADENOTONSILLECTOMY MONOCYTE MODULATION IN CHILDREN
WITH OBSTRUCTIVE CHRONIC ADENOTONSILLITIS ................................................................. 81
(Muhardjo)

MEMORY: WHERE AND HOW MEMORIES STORED? ...................................................................... 86
(Mohammad Hanafi)

CEREBRAL MALARIA.
I. THE ROLE OF ADHESION MOLECULES IN ITS PATHOGENESIS ........................................ 94
(Sri Hidayati BS)

RECONSIDERATION OF EARLY CHILDHOOD VACCINATION (AN EPIDEMIOLOGICAL
STUDY ON RELATIONSHIPS BETWEEN VACCINATION AND AUTISM) ..................................... 102
(Fuad Amisari and Nur Mukarromah)

ERYTHROCYTE SEDIMENTATION RATE DETERMINATION IN CHILDHOOD ASTHMA
DUE TO HOUSE DUST ALLERGY .................................................................................................... 107
(Anyardto Hansono and Martono Tri Utomo)

COMPARISON OF MEAN TEARS EXAMINATION RESULTS OF SCHIRMER TEST I
USING WHATMAN PAPER NO. 91 AND NO. 41 IN NORMAL PEOPLE ............................................. 111
(Kowena Ghazali Hoesin)

Review Article and Clinical Experience:
PLEIOTROPIC PROPERTIES OF STATINS
(Potential Benefits of "PECA-GOMES" for the Elderly) ................................................................. 115
(Askandar Tjokroprawiro)

YOUTH PROFILE IN SOME SUBURBAN AREAS IN EAST JAVA
(PRELIMINARY SURVEY OF THE INDONESIAN YOUTH STATURE AT THE FIFTIETH
ANNIVERSARY OF INDONESIA) ...................................................................................................... 122
(Johan Tunomgor and Hari K Lasmono)

THE ROLE OF PROBIOTIC CONTAINING FORMULA TO THE COURSE OF DIARRHEA ........ 127
(Reza G Ranuh et al)

Abstract ........................................................................................................................................... 132

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# Table of Contents

<table>
<thead>
<tr>
<th>No.</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>EDITORIAL Vol 39 No 2 2003</td>
<td>73 - 73</td>
</tr>
<tr>
<td>2</td>
<td>OPINION: CAN OR SHOULD HUMANS BE CLONED?</td>
<td>74 - 75</td>
</tr>
<tr>
<td>3</td>
<td>Correlations Between Psychological Stressor and Severe Preeclampsia (SPE)</td>
<td>76 - 80</td>
</tr>
<tr>
<td>4</td>
<td>Post-Adenotonsillectomy Monocyte Modulation in Children with Obstructive Chronic Adenotonsillitis</td>
<td>81 - 85</td>
</tr>
<tr>
<td>5</td>
<td>Memory: Where and How Memories Stored?</td>
<td>86 - 93</td>
</tr>
<tr>
<td>6</td>
<td>Cerebral Malaria I. The Role of Adhesion Molecules in Its Pathogenesis</td>
<td>94 - 101</td>
</tr>
<tr>
<td>7</td>
<td>Reconsideration Of Early Childhood Vaccination (an Epidemiological Study on Relationships Between Vaccination and Autism)</td>
<td>102 - 106</td>
</tr>
<tr>
<td>8</td>
<td>Erythrocyte Sedimentation Rate Determination in Childhood Asthma Due to House Dust Allergy</td>
<td>107 - 110</td>
</tr>
<tr>
<td>9</td>
<td>Comparison of Mean Tears Examination Results of Schirmer Test I Using Whatman Paper No. 91 and No. 41 in Normal People</td>
<td>111 - 114</td>
</tr>
<tr>
<td>10</td>
<td>Review Article and Clinical Experience: Pleiotropic Properties of Statins (Potential Benefits of â€œPECA-GOMESâ€• for the Elderly)</td>
<td>115 - 121</td>
</tr>
<tr>
<td>11</td>
<td>Youth Profile in Some Suburban Areas In East Java (Preliminary Survey of The Indonesian Youth Stature at The Fiftieth Anniversary of Indonesia)</td>
<td>122 - 126</td>
</tr>
<tr>
<td>12</td>
<td>The Role of Probiotic Containing Formula to The Course of Diarrhea</td>
<td>127 - 131</td>
</tr>
</tbody>
</table>
Cerebral Malaria I. The Role of Adhesion Molecules in Its Pathogenesis

Abstract

Cerebral malaria (CM), a complication in Plasmodium falciparum infection, causes high mortality among children in malaria endemic areas, with coma as major clinical symptom. Conventional explanation for the pathogenesis of the coma in CM is obstruction of small blood vessels in the brain by parasitized red blood cells which are sequestered there, that cause inadequate blood supply causing hypoxia and ischemia. Sequestration of parasitized rbc is started with the adherence of p-rbc to the endothelial cells lining blood vessels called cytoadherence, a specific ligand-receptor process involving adhesion molecules expressed on the surface of vascular endothels (i.e. CD 36, thrombospondin, ICAM-1, VCAM-1, E-selectin, Chondroitin-4-sulfate) and molecules on the surface of p-rbc which are related to the infecting parasites (e.g., Pf-EMP-1, Pf-HRP-1), followed or enhanced by rosette formation of normal rbc around p-rbc. Rosetting could involve similar p-rbc molecules involved in cytoadherence and facilitated by several serum proteins. The expression of adhesion molecules on the surface of endothelial cells is stimulated by parasite antigens and up-regulated by cytokines especially TNF-α (and IFN-γ). Efforts have been done to search antibody and medicine that could prevent cytoadherence and/or disrupt rosetting, and to control the production and effects of TNF-α.

Keyword : cerebral, malaria, Plasmodium, falciparum, adhesion, molecules, sequestration,