RINGKASAN

Toxoplasma gondii adalah protozoa penyebab toxoplasmosis. Penyakit ini bersifat zoonosis. Pada wanita hamil dan ternak bunting menimbulkan kelainan kongenital dan abortus sedang pada penderita AIDS menyebabkan encefalitis (Dubey, 2002; Wyler, 1990). Menurut Ghaffar (2001) infeksi toxoplasmosis kongenital sekitar 1-5 anak dari tiap 1000 wanita hamil, dimana 5-10% abortus, 8-10% kerusakan otak dan mata yang serius dan 10-13% bayi akan mengalami gangguan penglihatan. Meskipun 58-70% lahir normal, tetapi setelah beberapa bulan sampai beberapa tahun menunjukkan gejala berupa: retardasi mental, kelainan mata ringan sampai buta, hidrosefalus dan tidak mampu belajar (Dupoy-Camet, 2002, Ghaffar, 2001). Perkiraan kerugian ekonomis akibat toxoplasmosis kongenital dipaparkan oleh Robert dan Frenkel (1990) sebagai berikut: beberapa negara kehilangan income per kapita berkisar $ 0,2-5.8 trilyun, biaya perawatan dan pendidikan penderita antara $ 116 juta sampai $ 2,8 trilyun dan biaya pengobatan kelainan mata antara $ 368 juta sampai $ 8,7 trilyun.

Selain menimbulkan masalah pada wanita hamil, infeksi *T. gondii* juga banyak menimbulkan masalah berupa kelainan patologis fetus dan abortus pada hewan ternak bunting. Infeksi *T. gondii* merupakan penyebab utama abortus kambing dan domba di beberapa negara termasuk Australia dan Amerika serikat (Dubey, 2002). Frekuensi kejadian abortus dan kematian fetus pada induk domba terinfeksi *T. gondii* cukup tinggi dan anak domba lahir hidup jarang terjadi (Duncanson *et al*., 2001). Menurut Dubey dan Kirkbrid (1990), 65% dari 1564 ekor domba positif toxoplasmosis dan lebih dari 25% mengalami abortus. Hal tersebut tentu secara ekonomis merugikan peternak dan pemenuhan akan kebutuhan protein hewani tidak tercapai.

Mengingat kerugian yang ditimbulkan cukup besar maka diperlukan usaha pengendalian dan pencegahan. Pencegahan dengan program skreening memerlukan biaya banyak sedangkan kerugian jauh lebih tinggi dibanding keuntungan yang diperoleh (Abholz, 1993; Holliman *et al*., 1995). Tindakan pengobatan tidak sepenuhnya efektif menurunkan angka penularan dan masih mempunyai peluang 25% (Sciammarella, 2001; Wallon *et al.* 1999). Untuk kesuksesan pengobatan dan pencegahan tentu diperlukan pengetahuan mengenai penyakit, penyebab, kondisi dan termasuk mekanisme imunopatogenesis toxoplasmosis pada saat kebuntingan.

Penelitian ini bertujuan untuk mengetahui Peningkatan Ekspresi Interferon-gamma (IFN-γ) terhadap Angka Penularan Kongenital pada mencit bunting yang diinfeksi dengan *T.gondii*.

Dalam penelitian ini menggunakan 60 ekor mencit betina umur 2 bulan yang dibagi dalam 6 kelompok, sebagai berikut:

Kelompok 1. mencit tidak bunting tidak diinfeksi *T. gondii*
Kelompok 2. mencit tidak bunting diinfeksi *T. gondii*
Kelompok 3. mencit bunting 4,5 hari tidak diinfeksi *T.gondii*
Kelompok 4. mencit bunting 4,5 hari diinfeksi *T.gondii*
Kelompok 5. mencit bunting 14,5 hari tidak diinfeksi *T.gondii*

Kelompok 6. mencit bunting 14,5 hari diinfeksi *T.gondii*

Dosis infeksi 20 kista *T. gondii* hasil isolasi dari otak ayam. Empat hari setelah infeksi mencit dikurbankan. Serum dites dengan ELISA untuk mengetahui produksi IFN-γ sistemik dan uterus dibuat preparat hitopatologis dengan pengecatan imunohistokimia untuk mengetahui produksi IFN-γ lokal. Juga dilakukan bloting baik dari serum dan plasenta. Penentuan angka penularan kongenital dengan metode Fux *et al*, (2001)

Rancang percobaan dengan pola faktorial 2x3 untuk pengaruh infeksi terhadap produksi IFN-γ sistemik dan pola 2x2 untuk produksi IFN-γ lokal.

Hasil penelitian menunjukkan:

1. Infeksi *T. gondiii* menginduksi produksi INF-γ sistemik dan lokal
2. Produksi IFN-γ dalam serum mencit tidak bunting lebih rendah dari mencit bunting
3. Umur kebuntingan saat mendapatkan infeksi tidak mempengaruhi produksi IFN-γ lokal dan sistemik.
4. Kadar IFN-γ dalam serum dan diplasenta tidak mempengaruhi angka penularan kongenital
5. Umur kebuntingan saat mendapatkan infeksi berpengaruh terhadap angka penularan kongenital

SUMMARY

Toxoplasma gondii is a protozoon parasite cause toxoplasmosis, which is zoonotic disease On the pregnant woman and animal cause congenital anomalies and abortus, on the AIDS patient caused encephalitis (Dubey, 2002; Wyler, 1990).

Congenital toxoplasmosis found on 1-5 children of every 1000 pregnant women, where 5-10% were abortus, 8-10% were brain damages and occuler damage seriously and 10-13% of babies got eye disorder.

Eventhough, there were 58-70% normal birth, but after a few months until a few years showed some syndrome such as mental retardation, an light eye abnormality until blinness, hydrocepahalus and cannot to study well (Dupoy-Camet, 2002, Ghaffar, 2001). The prediction of economical decreased caused by congenitl toxoplasmosis according to Robert dan Frenkel (1990) such as folowed : some countries lost income per capita around $ 0,2-5.8 trillions, the care fore and patient education payment were $ 116 millions - $ 2,8 trillions and the fore of eye abnormalities $ 368 millions - $ 8,7 trillions.

The pathological fetus and abortus were another problems caused by toxoplasmosis on animal husbandary. Toxoplasmosis caused the main problem of abortus in sheep and goats in some countries included Australia and USA (Dubey, 2002). The frequency of abortus and fetal death on sheep females were high and the life kids birth were rarely happened (Duncanson *et al.*, 2001). According to Dubey dan Kirkbrid (1990), 65% of 1564 sheep were positive toxoplasmosis and more than 25% were abortus. All of those thing were harmfull for the farmers in order to prepare animals protein needs to reache it.

Because of large enaough in causing damages on human nd animals, there are some efforts need to do to control nd protect the creatures of this disease. The screening program to protect it needs much money, nevertheless, the damages were higher compared with the added values (Abholz, 1993; Holliman *et al.*, 1995). Medicated actions were not completely effective to decrease the dissemination of disease and were still 25% oportunity infection (Sciammarella, 2001; Wallon *et al.* 1999). Successful in medication and protection need the knowledge of disease, caused, condition and mechanism of toxoplasmosis imunopathogenesis pregnant condition..

An experimental reserch on this purpose were conducted in " Increasing Interferon-gamma (IFN-γ) expression and congenital dissemination number on *T. gondii* infection mice "

In this reserch were needs 60 female mice of 2 months old which were devided into six groups of treatment, wich were :

1 st group non pregnant female mice non infectin
2 nd group infection non pregnant female mice
3 rd group : 4,5 days pregnant mice non infection.
4 th group : infected 4,5 days pregnant mice.
5 th group : 14,5 days pregnant mice non infection
6 th group : infected 14,5 days pregnant mice.

Infection dose were 20 cysts of chicken isolated *T. gondii.* Four days after infection, the animals were sacrified, the sera were collected, the uterus organ were embedded on parafine. The sera were tested for IFN-γ systemic using ELISA and the uterus were tested for immunohistochemistry to get the IFN-γ local picture. On the other hand some part of uterus were tested by immunoblot analysis. Congenital dissemination were tested using Fux *et al*, technigue (2001)

Factorial program experimental design plants 2 x 3 for the influence of infection to the INF-γ production in sera; and 2 x 2 for the influence of .

The result of this research reveled that.

1. *T. gondii* infection indused systemic and local IFN-γ production.
2. IFN-γ production in serum of infected non pregnant mice less than infected pregnant mice.
3. The pregnant old when it were infected, have no influence on systemic nd local INF-γ production.
4. IFN-γ concentration in serum and local decidua don't influence the number of infection dissemination *T. gondii* to fetus.
5. The pregnant old when it was infected influenced the number of fetal dissemination infection.