Comparative Effectiveness Pyriproxyfen and Methophrene to Aedes aegypti Larvae from West Banjarmasin

by Istiana Istiana

Submission date: 18-Nov-2020 03:06PM (UTC+0700)
Submission ID: 1449855460
File name: nd_Methophrene_to_Aedes_aegypti_Larvae_from_West_Banjarmasin.pdf (13.04M)
Word count: 3280
Character count: 16611

PROCEEDING

international seminar

INTEGRATED VECTOR MANAGEMENT

Health and Environmental Perspectives

Saturday, October 26 th 2013
at Patra Jasa Semarang Convention Hotel
Semarang, Indonesia

Editor
Praba Ginandjar
Dina Rahayuning Pangestuti
Lintang Dian Saraswati

PUBLIC HEALTH FACULTY
DIPONEGORO UNIVERSITY

ISBN 978-602-097-389-0

COMPARATIVE EFFECTIVENESS OF PYRIPROXYFEN AND METHOPRENE TO *Aedes aegypti* LARVAE FROM WEST BANJARMASIN

Istiana
Departement of Parasitology, Medical Faculty of Lambung Mangkurat University
Correspondence: istiana_aribudi@yahoo.com

ABSTRACT

Background: Current larvicide used to control *Aedes aegypti* is temephos, although some studies have reported the presence of resistance. WHO recommended new larvicides contain active pyriproxyfen and methoprene from Insect growth Regulator (IGR) group as a substitute of temephos. This study aimed to determine the effectiveness pyriproxyfen and methoprene as larvicides and the time required in killig 50% larvae of *Aedes aegypti* from West Banjarmasin.

Method: This was an experimental study with post test only with control group design. Doses of pyriproxyfen were 0.5, 0.1, 0.05, 0.01, 0.008, and 0.005 ppm. Doses of methoprene were 0.01, 0.05, 0.1, 0.5, 1, and 1.5 ppm. Each treatment group was exposed to *Aedes aegypti* larvae. Larval metamorphoses and mortality were followed for 15 days. Analysis data used independent t test and probit.

Results: The result showed that pyriproxyfen effectively killed the larvae by 70% after seven days of exposure, while methoprene kills 50% in eight days. The optimum concentration to kill 50% of larvae (LC_{50}) for pyriproxyfen was 1.009ppm, while methoprene was 1.34 ppm. Test results of statistical analysis with Independent T test showed a significance different in killing larvae between pyriproxyfen and mehoprene (p=0.039).

Conclusion: Pyriproxyfen is more effective than Methoprene in killing larvae of *Aedes aegypti* from West Banjarmasin.

Keywords: effectiveness, pyriproxyfen, methoprene, *Aedes aegypti*

INTRODUCTION

Dengue Hemorrhagic fever (DHF) is an infectious disease that is still a public health problem in Indonesia, even likely to become endemic [1]. The case of DHF in 2012 is 90.245 cases recorded with the deaths of as many as 900 people. In South Kalimantan, the case of DHF is 1216 cases with the number of patients who died reached 19 cases (case fatality rate/CFR 1.5%). This number increased 3 fold compared to the year 2011 which is just 400 cases with the number of deaths are 7 people. Banjarmasin is the third most case with 56 cases. However, the cases who died reached 3 persons (CFR 5.3%) [2].

Dengue Haemorrhagic fever can only be controlled with vector eradication because the drug and vaccine for it has not found [1]. Control of the DHF can be done by eradicating adult and larvae of *Aedes aegypti* [3,4]. The adult mosquito eradication can be done by fogging and eradication of larvae can be done with *GERAKAN 3M* plus and using abate powder. However, long-term use of temephos has lower sensitivity against larvae [5-7]. The research of Gafur *et al* in North Banjarmasin showed a

decrease susceptibility of larvae to temefos [8]. Istiana *et al* also mentions that in West Banjarmasin showed a decrease in the susceptibility of larvae of *Aedes aegypti* to abate powder [9].

World Health Organization (WHO) has recommended the use of other insecticides as an alternative in dengue endemic areas that have resistance for temephos or have used temephos in the long time. One of the WHO recommendations is the use of Insect Growth Regulator (IGR) such as methoprene and pyriproxyfen [10].

The purpose of this study was to determine the effectiveness of larvicides pyriproxyfen and methoprene against *Aedes aegypti* larvae in the district of West Banjarmasin, South Kalimantan. This study is expected to provide scientific information as well as the basic for policy implementation on mosquito control program in Banjarmasin.

METHOD

This was an experimental study with Posttest-Only with Control Group Design. Subjects were larvae of *Aedes aegypti* Instar III-IV in second generation of colonization in Parasitology Laboratory, Medical Faculty of Unlam Banjarmasin. Independent variable wass Pyriproxyfen dose of 0.5 ppm, 0.1 ppm, 0.05 ppm, 0.01 ppm, 0.008 ppm, 0.005 ppm. While Methoprene dose was 0.01 ppm, 0.05 ppm, 0.1 ppm, 0.5 ppm, 1 ppm, and 1.5 ppm. Dependent variable was the *Aedes aegypti* larval mortality after administration of larvicides was followed for 15 days. Procedures used in this study was in accordance with the method set by WHO for experiments in the laboratory and the data obtained were analyzed using Probit Analyze with SPSS.

RESULTS

Results of research to determine effectiveness of IGR (pyriproxyfen and methoprene) can be seen in the following figure. Observation of larvae mortality and morphogenesis performed for fifteen days to determine the time to reach 70% or more larval mortality.

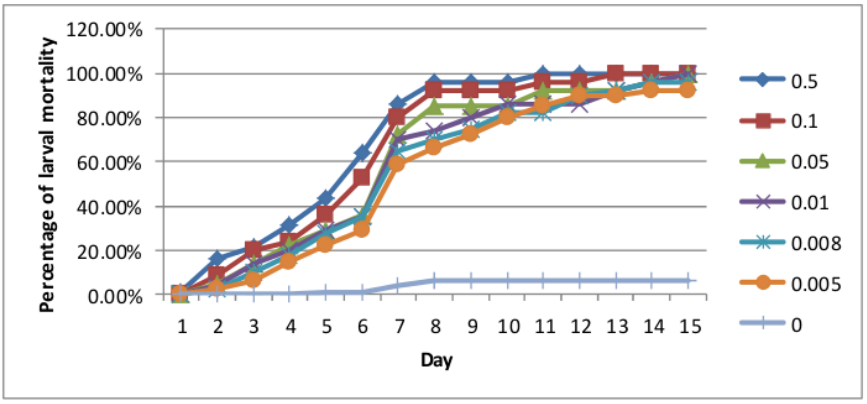

Figure 1. Percentage of Larvae of *Aedes aegypti*Mortalityfrom West Banjarmasinafter ExposuretoPyriproxyfen.

Figure 1 showed that the average percentage of dead larvae in the control group at 0% until day 4, and only 6.25% until day 15. This means development of mosquitoes in control group was well enough that larvae can develop into pupae and adult mosquitoes. The percentage of deaths in control group showed that deaths in other groups was purely caused by given larvicides. Because in this study the control larvae showed the average percentage mortality after 24 hours of exposure is 0% or no more than 20%, then itd oes not need to be corrected with Abbot Formula [11].

Statistical analysis by Kruskal Wallis test showed that all groups of dosage were differences in larval mortality (p=0.019). This is according to tests performed by Miller et.al. who also showed significant difference of larval mortality in the dosage of groups (p=0.001) [10]. The result of Mann-Whitney test to determine dose groups had significant differences, indicated that all groups were different (p<0.05). This significant difference can be seen in the 0.5 ppm dose group with the control group (p=0.001), 0.1 ppm with control (p=0.005), 0.05 ppm with controls (p=0.007), 0.01 ppm with controls (p=0.007), 0.008 ppm with control (p=0.010), and 0.005 ppm with controls (p=0.010). Subsequent data analysis using probit analysis to determine the dose required to obtain 50 % mortality and 90 % of the test larvae as in Table 1.

Table 1. Probit analysis results for Dose Required to Kill 50 % (LC 50) and 90 % (LC 90) Larvae Test

Larval mortality (%)	Doses (ppm)
10	0,314
20	0,553
30	0,735
40	0,872
50	**1,009**
60	1,146
70	1,294
80	1,466
90	**1,704**
95	1,901
99	2,271

The results with various concentrations of the administration methoprene to *Aedes aegypti* larvae are shown in Figure 2. It shows that in the control group, the average percentage of larvae on the eighth day mortality of 6.25 % to 7.5 % on day 15, so it does not need to be corrected using the formula abbott. Abbot formula is used if the percentage of control group mortality of more than 20 % in 24 hours of observation.

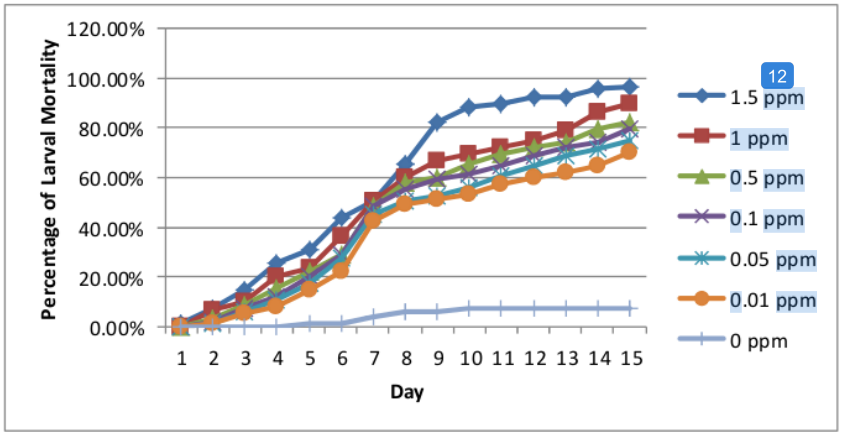

Figure 2. Percentage of Larvae of *Aedes aegypti*Mortalityfrom West Banjarmasin after Exposureto Methoprene.

Analyzed with Kruskal Wallis test showed the results of p=0.040, which means that there are significant differences between treatment groups. Further to determine significant differences between treatment groups, using the Mann-Whitney test. The results obtained indicate that there are several groups of p<0.05. This means that there are significant differences in the treatment groups between the control group with the group with a concentration of 0.05 ppm, 0.1 ppm, 0.5 ppm, 1 ppm and 1.5 ppm.

Probit analysis is then performed to determine the dose required to kill 50 % and 90 % larval test with results as shown in Table 2.

Table 2. Probit analysis results for Methophren Dose required Killing 50 % (LC 50) and 90 % (LC 90) Larvae of *Aedes aegypti*

Larval mortality (%)	Doses (ppm)
10	0,445
20	0,675
30	0,835
40	0,997
50	**1,342**
60	1,461
70	1,954
80	2,257
90	**2,704**
95	2,901
99	3,178

It shows that the LC50 for Methophren in this study was 1.342 ppm and the LC90 is 2.704 ppm. It means to kill larvae by 50 % and 90 % of the required dose.

DISCUSSION

Figure 1 shows that not all doses tested had an average percentage of larval mortality trials more than 70 % after 7 days, whereas at the highest dose of 0.5 ppm and 0.1 ppm indicate the average percentage of larval mortality trials reached more of 80 % and 86%. In the last days, all dose shows the average percentage of dead larvae reached more of 90% and for dose of 0.5 ppm and 0.1 ppm reached 100%. These results indicate that the percentage of larval mortality increased along with concentration or dose of larvicide and the length of time observation increased.

Based on the calculation criteria for larvicidal efficacy, as an active ingredient pyriproxyfen, after 24 hours of observation, it showed that all doses tested did not meet the criteria of efficacy due to larval mortality rate is below 70 % [12]. This is because the nature of active ingredient pyriproxyfen does not kill larvae directly, but inhibits development of larvae into pupae and adult mosquitoes. Pyriproxyfen that is in the water easily penetrates the skin mosquito larvae then enter into haemolymph. Pyriproxyfen in the haemolymph caused corpusallatum not produce juvenile hormone which caused the larvae do not develop into pupa or mosquitoes [13]. This was proven by observation until day 15, at all doses tested there was no change in the development of larvae into adult mosquitoes. This is very different from the control groups where the most of larvae change into adult mosquitoes.

Result of this study have similarities with research conducted by Munif, where the IGRA deal with active ingredient Pyriproxyfen, at dosage of 0.01 ppm has ability to inhibit growth of larva into mosquitoes reached 100 % after 1 week of exposure [14]. Although in this study dosage of 0.5 ppm inhibited 100 % of larvae growth on day 11, and at dosage 0.1 ppm on day 13.

Table 1 reveals that the dose required killing 50% of larvae of *Aedes aegypti* in this study was 1,009 ppm, while the dose required to kill 90% of test larvae was 1,704 ppm. Pyriproxyfen has a unique pattern of activity and influence on physiology, morphogenesis, reproduction, and especially insect embryogenesis metamorphosis that has properties such as *Aedes aegypti* mosquito is the primary vector of dengue fever [13]. Although larvicidal active ingredient pyriproxyfen takes time to kill mosquito larvae and pupae, pyriproxyfen has a long residual life according to tests performed by the Munif [14], at dosage 0.01 ppm pyriproxyfen can inhibit the growth of *Aedes aegypti* larvae into adult mosquitoes by 100% until fifth of week.

Percentage of larval mortality at various doses methoprene test increased with increasing concentrations of the test. This is showed in figure 2. Based on the results of the study, the average percentage rate larval mortality during the 24 hour at 0.41 % due to death on the first day it just occurred in the group treated with a dose of 1.5 ppm. On the eighth day of observation, the average percentage of larval mortality test all doses of more than 50 % except at doses of 0.01 ppm, and the highest concentrations of his death only 65 %. This percentage increased with the length of observation time, where at day 15, the average mortality in all treatment groups of more than 70 %, and reached 96.5 % at the 1.5 ppm dose group.

Increased mortality in line with the length of time due to the nature of the active ingredient methoprene kills larvae not directly, but inhibits the development and cause the death of the pupal stage and growth failure of adult mosquito's stage. This is according to research conducted Braga et al that the greatest number of percentage

mortality of larvae fed methoprene, found in the pupa stage, not on stage of larvae [15]. The same thing also expressed by Wu et al, that the increasing concentration of methoprene the percentage pupal mortality also increased [16].

However, because this is the nature of larvicides as a barrier to development, so that the higher concentration is given, then the percentage of deaths would be even greater. This is supported by Braga et.al. study [17], Wu et.al [16] and Pranoto [18] which shows percentage pupal mortality reached 100 % in the third week after treatment. It can support that methoprene inhibit larval development and kills pupa stage. Because on the fifth day, the control group there were a lot of mosquitoes mature and grow upon the eighth day. However, none of the treatment groups of larvae into adult mosquitoes up to eight days.

Methoprene is a larvicide that serve as artificial hormones which regulate growth-stage larvae (IGR-Insect Growth Regulators). Mechanism of action of this hormone does not kill larvae directly but inhibits the formation cytine during larval grow than disrupt the process of shape change into pupae and mosquitoes (inhibitor development), so it cannot grow into pupae of mosquitoes or mosquitoes can grow into, but not normal [19].

To compare the effectiveness of pyriproxyfen with methoprene performed by independen t Ttest analysis. The analysis showed p=0.039, which means that there is a significant difference between the effects larvicides produced by pyriproxyfen with methoprene. If seen from the LC50 and LC90 between pyriproxyfen with methoprene, it appears that pyriproxyfen better than methoprene.

In the life cycle of the mosquito phase of growth the reare egg, larva, pupa and adult. This growth phase requires growth regulator hormone called juvenile hormone, an enzyme that plays a role influencing responsibility to determine character influence larvae even in the nucleus and modification ecdysis effects that determine the process of change in the cuticle and the turn instar stage. The role of the hormone is then applied to the artificial hormone pyriproxyfen and methoprene. IGR is a hormone that is controlling the growth of biorational control, which is a process that affects the growth of larva-pupa to adult ratio by regulating hormonal biology itself larvae that affect physiology of morphogenesis in the larval-pupal stage that will shape the mosquito pupa and disabilities resulting from the furnishing inexcessive dosage [20].

Larvae that survive will grow into pupae, but there has been a maturation and cellular differentiation thus become abnormal growth pupa. Larvae undergo ecdysis, the replacement process is not complete because the cuticle skint hat should be separated from the body of the larvae turned out to cohere in the posterior, thus masking tool that works spiracle respiration, this result can not breathing pre-pupae and finally die. In the pupa stage, juvenile hormone does not work anymore, but the hormone active ecdyson serves to form organs, organs and organ systems respiration [21].

In general, IGR can give the effect of long-term survival (3 to 6 months) with an appropriate dose. This is consistent with research Pranoto that shows the percentage of deaths pupa until the fourteenth week amounted to 99.25%. So that repetition of administration can be done for 3 months [18].

CONCLUSIONS

Effective dose of Pyriproxyfen to kill 50 % of larvae of *Aedes aegypti* is 1.009 ppm, while to kill 90 % larvae is 1.704 ppm. Effective dose of Methoprene to kill 50 % of larvae of *Aedes aegypti* is 1.342 ppm and effectively kill 90 % at a dose of 2.704 ppm. Pyriproxyfen have better effectiveness in killing the larvae of *Aedes aegypti* compared with Methophren.

Further research needs to be done to determine the effectiveness pyriproxyfen and methophren in the field so that it can be applied directly in the community, and ultimately can help reduce morbidity and mortality due to dengue fever.

REFERENCES

1. Mansjoer A, 2001. Demam Dengue. Dalam Ilmu Penyakit Dalam Infeksi Trofik. Edisi 3 Jilid 1. Jakarta, Media Aesculapius,
2. Dinas Kesehatan Provinsi Kalimantan Selatan. 2012 Profil Kesehatan Provinsi Kalimantan Selatan Tahun 2011.
3. Krueger. 2006. Effective Control of Dengue Vectors With Curtains and Water Container Cover Treated With Insecticide in Mexico and Venezuela: Cluster Randomized Tri[illegible] *BMJ* 75: 1247-52
4. Zulhasril dan Frieda FB. Penggunaan piretroid sintetik (permetrin) dalam pengendalian larva vector demam berdarah dengue (Aedes aegypti L). J Kedokt Yarsi 2006;14(1): 29-33
5. Sungkar, S., Zulhasril. 1997. Status kerentanan larva *Aedes aegypti* terhadap temefos di beberapa daerah di Jakarta. *Majalah Kedokteran Indonesia* 47: 25-28.
6. de Carvalho, M.D.S., Caldas, E.D., Degallier, N., Vilarinhos, P.D.T., de Souza, L.,Amelia, M., Yoshizawa, C., Knox, M.B., de Oliveira, C. 2004. Susceptibility of *Aedes aegypti* larvae to the insecticide temephos in the Federal District, Brazil. *Revista De Saude Publica* 38: 623-629.
7. Rodriguez, M.M., Bisset, J., Ruiz, M., Soca, A. 2002. Cross-resistance to pyrethroid and organophosphorus insecticides induced by selection with temephos in *Aedes aegypti* (Diptera : Culicidae) from Cuba. *Journal of Medical Entomology* 39: 882-888.
8. Gafur A, Mahrina, Hardiansyah. 2006. Kerentanan larva Aedes aegypti dari Banjarmasin Utara. *Bioscientiae* 3(2) : 73-82
9. Istiana, Heriyani F, Isnaini. 2012. Status Kerentanan larva Aedes Aegypti terhadap Temephos di Banjarmasin Barat. Jurnal Buski 4(2) : 1-6.
10. WHO, CDS, WHOPES, GCDPP. 2005. Guidelines for Laboratory And field Testing of Mosquito Larvicides.
11. Rodriguez, M.M., Bisset, J., De Fernandez, D.M., Lauzan, L., Soca, A. 2001. Detection of insecticide resistance in *Aedes aegypti* (Diptera : Culicidae) from Cuba and Venezuela. *Journal of Medical Entomology* 38: 623-628.
12. Campos, J., Andrade, C.F.S. 2001. Larval susceptibility to chemical insecticides of two *Aedes aegypti* populations. *Revista De Saude Publica* 35: 232-236.
13. Andrighetti MTM, Cerone F, Rigueti M, Galvani KC, da-Gracia-Macoris MdL. 2008. Effect of pyriproxyfen in *Aedes aegypti* populations with different levels of susceptibility to the organophosphate temephos. Dengue Bulletin 32 : 186-198.

14. Munif A. Pengaruh residu pyriproksifen 0,5% terhadap pertumbuhan larva aedes aegypti pada berbagai simulasi wadah air formula tepung pada berbagai instar larva Aedes aegypti di laboratorium. CDK 1997;119(2) : 42-9
15. Braga IA, Mello CB, Peixoto AA, Valle D. Evaluation of methoprene effect on Aedes aegypti (Diptera : Cullicidae) development in laboratory conditions. Mem Inst Oswaldo Cruz, Rio de Janeiro 2005: 100(4) : 435-40
16. Wu Y, Parthasaraty R, Bai H, Palli SR. Mechanisme of midgets remodelling : juvenile hormone analog methoprene block midgut methamorphosis by modulating ecdysone action. Mechanisme of development 2006;123(7) : 537 – 40
17. Braga IA, et al.2005. Effectiveness of methoprene, an insect growth regulator, agains temephos resistant Aedes aegypti populations from different Braziliense localities, under laboratory condition. J Med Entomol 42: 830-837
18. Pranoto. 1994. Pengaruh Insect Growth Regulator (IGR) Altosid 1z,3G terhadap populasi Larva nyamuk Aedes aegypti linneus. Buletin Penelitian Kesehatan 1994;22(4):1-9
19. Maharani A, Handayani FD. Insect growth regulator (IGR) terhadap larva Aedes aegypti di laboratorium. Profesi Medika 2005;5(2):67-75
20. Kamal HA, Khater El. 2010. The biological effect of insect growth regulators : pyriproxyfen and diflubenzuron on the mosquito Aedes aegypti. J Egypt Soc Parasitol.40(3):565-74
21. Ponlawat, A., Scott, J.G., Harrington, L.C. 2005. Insecticide susceptibility of *Aedes aegypti* and *Aedes albopictus*across Thailand. *Journal of Medical Entomology* 42: 821-825.

Comparative Effectiveness Pyriproxyfen and Methophrene to Aedes aegypti Larvae from West Banjarmasin

ORIGINALITY REPORT

14%	10%	9%	6%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	L. FACCHINELLI. "Development of a novel sticky trap for container-breeding mosquitoes and evaluation of its sampling properties to monitor urban populations of Aedes albopictus", Medical and Veterinary Entomology, 6/2007 Publication	1%
2	Submitted to University of Nebraska, Lincoln Student Paper	1%
3	Submitted to University of Durham Student Paper	1%
4	hdl.handle.net Internet Source	1%
5	www.cabi.org Internet Source	1%
6	Rocha, L.F.N., N.A. Sousa, J. Rodrigues, A.M.L. Catão, C.S. Marques, É.K.K. Fernandes, and C. Luz. "Efficacy of Tolypocladium cylindrosporum against Aedes aegypti eggs, larvae and adults",	1%

Journal of Applied Microbiology, 2015.
Publication

7	Belinato, Thiago Affonso, Ademir Jesus Martins, José Bento Lima, and Denise Valle. "Effect of triflumuron, a chitin synthesis inhibitor, on Aedes aegypti, Aedes albopictus and Culex quinquefasciatus under laboratory conditions", Parasites & Vectors, 2013. Publication	1%
8	www.kalbefarma.com Internet Source	1%
9	core.ac.uk Internet Source	1%
10	Kara K. Ballenger-Browning. "Multi-modal *Aedes aegypti* mosquito reduction interventions and dengue fever prevention", Tropical Medicine & International Health, 12/2009 Publication	1%
11	Ling Tian, Enen Guo, Yupu Diao, Shun Zhou, Qin Peng, Yang Cao, Erjun Ling, Sheng Li. "Genome-wide regulation of innate immunity by juvenile hormone and 20-hydroxyecdysone in the Bombyx fat body", BMC Genomics, 2010 Publication	1%
12	J.C. Hilborn, N. Quickert. "Evaluation of the Byron Model 233A Air Quality Analyzer", Instrumentation Science & Technology, 2008	1%

	Publication	
13	id.erudit.org Internet Source	1%
14	fexdoc.com Internet Source	1%
15	www.lontar.ui.ac.id Internet Source	1%
16	journal.unnes.ac.id Internet Source	1%
17	www.researchgate.net Internet Source	<1%
18	id.scribd.com Internet Source	<1%
19	pt.scribd.com Internet Source	<1%
20	ejurnal-citrakeperawatan.com Internet Source	<1%
21	www.ijeco.org Internet Source	<1%
22	journal.entsocbc.ca Internet Source	<1%
23	www.neliti.com Internet Source	<1%

24	Submitted to ku Student Paper	<1%
25	ir.lib.hiroshima-u.ac.jp Internet Source	<1%

Exclude quotes	On	Exclude matches	< 10 words
Exclude bibliography	Off		