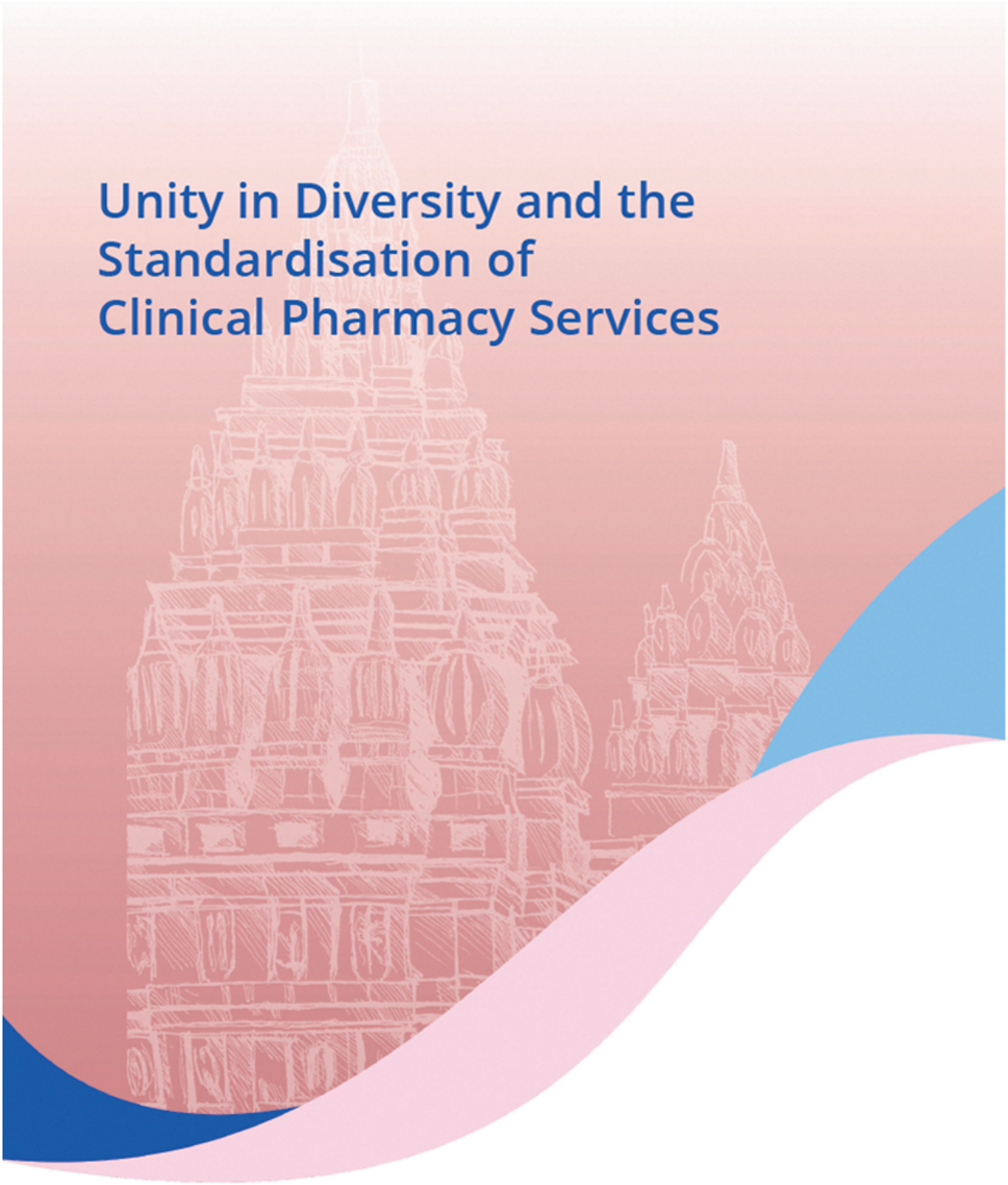
Unity in Diversity and the Standardisation of Clinical Pharmacy Services



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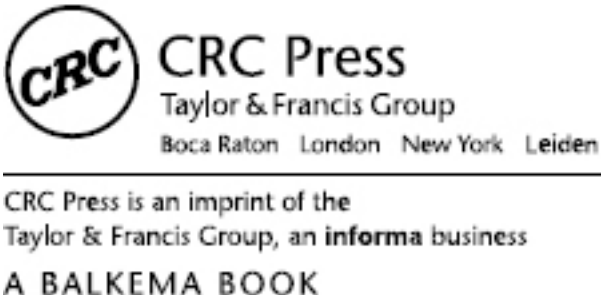

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Preface

The original idea of ACCP came from Asian pharmacists who were looking for a practical conference at which they could exchange and share ideas on the concept of clinical pharmacy. In 1996, representatives from China, Korea, Japan, and USA met in Seoul, Korea to plan for the first conference. As a result, the first East Asia Conference on Developing Clinical Pharmacy Practice and Clinical Pharmacy Education (EACDCPPE) was held in America in 1997. Only 36 representatives attended and pioneers planned it as bi-annual meeting.

In 1999, the second EACDCPPE was successively held in Shanghai. This conference enabled more representatives in Asian countries to realize the differences between Asian and Western countries in the development of clinical pharmacy. When the third conference was held in Japan in 2003, the title of the conference was changed to Asian Conference on Clinical Pharmacy (ACCP). This opened the conference to more Asian countries; also the subject of clinical pharmacy was more strengthened. With a series of other Asian countries such as Philippines, Indonesia, Singapore, and so on attending ACCP, as well as with the rapid development of clinical pharmacy in Asia, every country was enthusiastic about attending and holding this conference. At the 5th conference in Malaysia in 2005, the decision was made among the representatives of the member countries to hold the conference annually instead of biannually for efficiency and convenience in regard to communicating and sharing about clinical pharmacy.

During the past 20 years, ACCP has been a major event in the clinical pharmacy scope in Asia and has been conducted in various countries especially in Asia. Clinical pharmacists have attended this prestigious meeting to share their experience in the fields of practice, research, and education on clinical pharmacy. Clinical pharmacist experts from USA, Canada, Australia, and UK have continuously come to transfer their knowledge and shared advance clinical pharmacy practice experiences. This conference supports rapid knowledge and experience transfer and enhances the emergence of clinical pharmacy practice in Asia.

Indonesia hosted the 8th ACCP in Surabaya in 2008, and again this year Indonesia has successfully hosted the 17th ACCP in Yogyakarta from 28th to 30th July 2017. This year's conference was also a celebration of 20 years of ACCP with the theme "Unity in Diversity and the Standardisation of Clinical Pharmacy Services." At ACCP 2017, there were 6 preconference workshops, poster sessions consisted of 199 posters, 21 oral presentation sessions consisted of a total of 142 oral presentations, and there were symposiums with 47 speakers, 2 plenary sessions with 4 speakers and 4 keynote speeches regarding various current issues in clinical pharmacy. About 1,133 participants attended the conference from 16 different countries.

This ACCP 2017 proceeding provides an opportunity for readers to engage with selected papers presented at the 17th ACCP 2017. This book is also a valuable contribution to gaining a better understanding about the development of clinical pharmacy particularly in Asian countries and the future global challenges. Readers will find a broad range of research reports on topics of clinical pharmacy, social and administrative pharmacy, pharmacy education, pharmacoeconomics, pharmacoepidemiology and other topics in pharmacy. The readers will also discover both common challenges and creative solutions emerging from diverse settings in developing clinical pharmacy services.

The editors would like to thank all those who have contributed to submit full papers for this 17th ACCP conference. We received 119 papers from the conference and after a rigorous peer-review, 68 papers were accepted for publication in this proceeding of which 56 are from Indonesia and 12 from Australia, Malaysia, the Philippines, and Thailand. We would like to express our special appreciation and sincere thanks to the scientific committee and the reviewers who have selected and reviewed the papers, and also the technical editor's team (Ms Arie Sulistyarini and Ms Muffarihah) who helped carry out the page layout and check the

consistency of the papers with the publisher's template. It is a great honour to publish selected papers in this proceeding by CRC Press/Balkema (Taylor & Francis Group). Our special gratitude goes to the steering committee, the chairman of the conference and the members of the organizing committee involved in preparing and organizing the conference. Finally, we would like to thank Universitas Airlangga, Indonesian Pharmacist Association, Universitas Gadjah Mada, Universitas Ahmad Dahlan, Universitas Islam Indonesia, Universitas Muhammadiyah Yogyakarta and Universitas Sanata Dharma for their endless support during the conference. Last, but not least, we also place on record our sense of gratitude to one and all who, directly or indirectly, have lent a helping hand to this conference.

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Efficacy of honey vinegar in hyperlipidemic rats (*Rattus norvegicus*)

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ABSTRACT: Honey vinegar is a well-known traditional medicine used for preventing hyperlipidemia. Therefore, this study aimed to prove the efficacy of honey vinegar as an alternative herbal medicine to treat hyperlipidemia in rats (*Rattus norvegicus*). A total of 30 rats were divided into three groups: negative control, positive control, and test groups. The negative control group was given demineralized water. The positive control group was given fried oil (4 ml/kg BW) and pork oil (5 ml/kg BW) orally. The test group was given fried oil (4 ml/kg BW), pork oil (5 ml/kg BW), and honey vinegar (10 ml/kg BW) orally. Each group underwent the respective therapy for 14 days. On the 15th day, the LDL-cholesterol, HDL-cholesterol, triglyceride, and total cholesterol levels of all the groups were tested. The results demonstrated that the test group treated with honey vinegar had lower cholesterol levels ($P < 0.05$). Therefore, it can be concluded that honey vinegar has good efficacy in reducing hypercholesterolemia in rats.

1 INTRODUCTION

An abnormal blood lipid profile may be an initial symptom of arterosclerosis. The condition of having a persistently high blood lipid profile is known as hyperlipidemia. Long-term hyperlipidemia is one of the main risk factors for several medical problems such as Cardiovascular Disease (CVD), diabetes mellitus, hypertension, and stroke (Dera-khshandeh-Rishehri et al. 2014). The increase in the prevalence of these diseases globally is related to the unhealthy lifestyles and diets of most people in developed and developing countries, including Indonesia (Indonesian Health Ministry 2013).

Exploring the beneficial effects of natural ingredients has an important role to play in developing a new complementary medicine to optimize the prevention or treatment of hyperlipidemia (Derakhshandeh-Rishehrier et al. 2014). Vinegar, a liquid consisting of natural ingredients, is derived from fruits and vegetables. It is one of the important sources of polyphenolic compounds. Polyphenolic compounds are well-known antioxidants that play an important role in preventing several CVD such as hypertension and heart attack (Budak et al. 2011 Soltan & Shehata 2012).

Honey, which is another type of natural product, is mainly made of fructose and glucose. Natural honey is also a well-known antioxidant. Research has demonstrated that natural honey is associated with weight loss and known to have beneficial effects on risk factors for CVD (Derakhshandeh-Rishehri et al. 2014). A recent study has reported that honey vinegar can decrease total cholesterol and HDL-cholesterol levels, but with no significant effect on LDL-cholesterol and triglyceride levels (Derakhshandeh-Rishehri et al. 2014). Therefore, this study aimed at proving the efficacy of honey vinegar in treating hyperlipidemia in rats (*Rattus norvegicus*).

2 METHODS

Honey vinegar obtained from a bee farm factory (Rima Raya) in Lawang, Indonesia was used in this study. The hypercholesterolemic rats (*Rattus norvegicus*) were daily administered with honey vinegar (10 ml/kg BW) orally for 14 days. A total of 30 healthy male Wistar rats (*Rattus norvegicus*), aged 2–3 months old and weighing 150–250 g, were used in this study. The rats were adapted for one week before the experiment, and divided into three groups. The negative control group was given demineralized water. The positive control group was given fried oil

(4 ml/kg BW) and pork oil (5 ml/kg BW) orally. The test group was given fried oil (4 ml/kg BW), pork oil (5 ml/kg BW), and honey vinegar (10 ml/kg BW) orally. Each group underwent the respective therapy for 14 days.

On the 15th day, the rats were killed after overnight fasting. A blood sample from each rat was collected in a tube. Serum was separated by centrifugation at 2000 rpm for 10 minutes and stored at –20°C until further analysis. The LDL-cholesterol, HDL-cholesterol, triglyceride, and total cholesterol levels of the rats were measured by using the enzymatic method at Center of Health Laboratory Surabaya, Indonesia. Data are expressed as mean ± SD. Results were analyzed and compared between each group using an independent T-test (SPSS version 17.0). $P < 0.05$ was considered as significant.

3 RESULTS AND DISCUSSION

The results demonstrated that the mean cholesterol levels of the negative control group were significantly different from those of the positive control group (negative control: total cholesterol 83.00 ± 7.35 mg/dl, HDL-C 47.60 ± 6.27 mg/ dl, LDL-C 22,30 ± 3.65 mg/dl and triglyceride 60.50 ± 14.38 mg/dl versus positive control: total cholesterol 104.50 ± 16.69 mg/dl, HDL-C 62.30 ± 4.97 mg/dl, LDL-C 28,40 ± 7.29 mg/dl and triglyceride 84.10 ± 17.75 mg/dl; $P < 0.05$). This difference can be attributed to the combined administration of fried oil and pork oil to the positive control group for 14 days. The result is also consistent with that reported by Buettner et al. (2007), showing that prolonged feeding with fat-rich diets can lead to an increase in body weight and lipid profile in susceptible rats in the range of 10% to 20% when compared with standard chowfed controls.

The test group had a significantly lower lipid profile than the positive control group (test group: mean total cholesterol level 68.90 ± 12.77 mg/dl, mean HDL-C 41.60 ± 1.39 mg/dl, mean LDL-C 19.00 ± 4.35 mg/dl and mean triglyceride 64.90 ± 19.63 mg/dl; $P < 0.05$). The results are summarized in Table 1.

The lipid profiles of the test group were decreased significantly because of honey vinegar. However, the underlying mechanism still remains elusive. Honey is a type of natural product that is mainly made of fructose and glucose. One theory states that honey vinegar can cause changes in lipid metabolism because of its high fructose level (Derakhshandeh-Rishehri et al. 2014). Fructose can reduce the activity of lipoprotein lipase (LPL) and lecithin cholesterol acyl transferase (LCAT). A significant reduction in LPL activity can prevent the hydrolysis of VLDL and chylomicrons, thereby reducing the synthesis of HDL-C, LDL-C, triglyceride and total cholesterol (P. Aurag & C.V. Anuradha, 2002).

LCAT also plays an important role in the pathway of HDL-C synthesis. Its reduced activity can result in the metabolism and synthesis of HDL-C, LDL-C, triglyceride and total cholesterol (Thirunavukkarasu & Anuradha 2004). The present results were consistent with that of Bahesti et al. 2012, who used apple cider vinegar. The authors showed that consumption of apple cider vinegar for 8 weeks reduced harmful lipids such as total cholesterol, LDL, and triglyceride, in hyperlipidemic individuals who did not use any lipid-lowering drugs. Another study conducted by Fushimi et al. reported a significant reduction in cholesterol and triglyceride levels when high cholesterol-fed rats consumed acetic acid. It was concluded that the acetic acid in vinegar can decrease the oxidation of fatty acids, inhibit lipogenesis in the liver, and thus decrease the concentrations of triglyceride and cholesterol.

Table 1. Lipid profiles of the negative control, positive control, and test groups.

Groups	Number of rats	Levels (mg/dL)			
		Total C*	HDL-C*	LDL-C*	TG*
Negative control	1	86	53	27	38
	2	85	48	21	64
	3	78	51	20	44
	4	77	52	16	50
	5	87	49	21	61
	6	73	44	18	79
	7	75	36	24	50
	8	83	38	26	68
	9	89	51	25	75
	10	97	54	25	76
	x ±	83.00	47.60	22.30	60.50
	SD	7.35	6.27	3.65	14.38
Positive control	1	105	64	30	80
	2	86	55	21	68
	3	109	65	30	73
	4	100	61	31	80
	5	97	64	22	112
	6	83	63	16	66
	7	103	53	29	109
	8	127	67	36	104
	9	137	69	41	82
	10	98	62	28	67
	x ±	104.50	62.30	28.40	84.10
	SD	16.69	4.97	7.29	17.75
Test group	1	95	59	26	38
	2	71	43	16	86
	3	69	43	18	50
	4	74	57	12	71
	5	78	54	20	62
	6	55	24	22	85
	7	63	25	24	67
	8	59	39	14	55
	9	74	52	18	95
	10	51	20	20	40
	x ±	68.90	41.60	19.00	64.90
	SD	12.77	14.39	4.36	19.63

* Significantly different ($P < 0.05$).

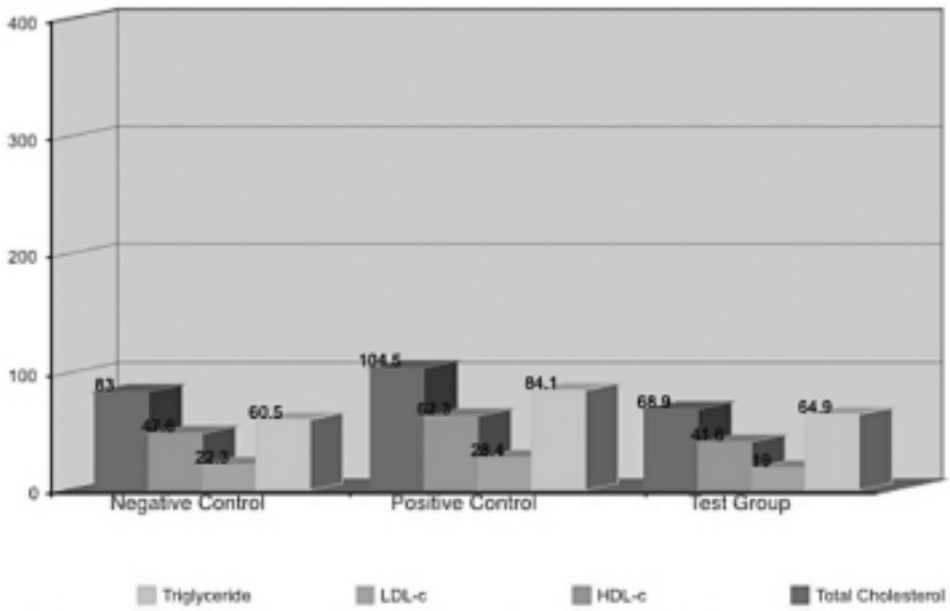

Figure 1. Lipid profiles of the negative control, positive control, and test groups.

4 CONCLUSION

Honey vinegar has shown to have good efficacy in reducing the levels of total cholesterol, HDL-C, LDL-C, and triglyceride in hyperlipidemic rats. Further studies on human subjects are needed to verify its efficacy in humans.

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