PSYCHO-SOCIAL CONSTRUCT CHARACTERIZATION OF ADOLESCENTS FOOD INSECURITY EXPERIENCE

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Contents

2. Letter of Offer (Research Grant)

Surat Kami : 600-RMI/RAGS 5/3 (121/2012)
Tarikh : 15 Disember 2012

Encik Nazrul Hadi Ismail
Fakulti Sains Kesihatan
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Tuan

KELULUSAN DANA PEMBUDAYAAN PENYELIDIKAN (RAGS) 2012

Tajuk Projek	:	*Psycho-social Construct Characterization of Adolescents Food Insecurity Experience*
Kod Projek	:	600-RMI/RAGS 5/3 (121/2012)
No. Rujukan Penaja	:	RAGS/2012/UITM/SKK10/5
Bidang	:	Sains Kesihatan dan Klinikal (Sains Kesihatan Bersekutu)
Tempoh	:	15 Disember 2012 - 14 Disember 2014 (24 bulan)
Peruntukan Diluluskan (KPT)	:	RM56,000.00
Peruntukan Pengurusan	:	RM 2,750.00 (5%)
Peruntukan Pengoperasian	:	RM52,250.00 (95%)
Ketua Projek	:	Encik Nazrul Hadi Ismail

Dengan hormatnya perkara di atas adalah dirujuk.

2. Sukacita dimaklumkan pihak Kementerian Pengajian Tinggi melalui surat JPT.S(BPKI)2000/018/02Jld.3(4) yang bertarikh 7 Disember 2012 telah meluluskan kertas cadangan penyelidikan tuan untuk dibiaya di bawah Dana Pembudayaan Penyelidikan (RAGS) 2012.

3. Bagi pihak Universiti kami mengucapkan tahniah kepada tuan kerana kejayaan ini dan seterusnya diharapkan berjaya menyiapkan projek ini dengan cemerlang.

4. Peruntukan kewangan akan disalurkan melalui tiga (3) peringkat berdasarkan kepada laporan kemajuan serta kewangan yang mencapai perbelanjaan lebih kurang 50% dari peruntukan yang diterima.

Peringkat Pertama	20%
Peringkat Kedua	40%
Peringkat Ketiga	40%

5. Untuk tujuan mengemaskini, pihak tuan adalah diminta untuk melengkapkan semula kertas cadangan penyelidikan, mengisi borang setuju terima projek penyelidikan dan menyusun perancangan semula bajet yang baru seperti yang diluluskan. Sila lihat lampiran bagi tatacara tambahan untuk pengurusan projek.

Sekian, harap maklum.

"SELAMAT MENJALANKAN PENYELIDIKAN DENGAN JAYANYA"

Yang benar

PROFESOR DR. ABU BAKAR ABDUL MAJEED
Penolong Naib Canselor (Penyelidikan)

Dilampirkan

3.

5.2 Enhanced Executive Summary

Food insecurity, the inability to have sufficient, safe and nutritious food for an active and healthy life, was found to be closely associated with adverse health outcomes. However, limited studies can be found that clearly explains lipid profile and inflammatory events among food secure and insecure individuals, especially among young adults in university, thus creating the need for further research. This study investigated both groups including their gender distribution to determine lipid profile such as total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) and inflammatory marker, high sensitivity C reactive protein (hs-CRP), with waist circumference (WC), fat mass index (FMI) and waist-to-height-ratio (WHTR). A comparative cross-sectional study was carried out among participants aged between 18 to 25 years old (N=124) who were selected through the Adults Food Security Survey Module (AFSSM) and participated in blood draw procedures. Well-established blood markers of lipid profile and inflammatory marker were measured. Percentage of food secure individuals (56.5%) was slightly higher than food insecure (43.5%). Although mean (M) of Hs-CRP for male and female (M=1.000, M=0.645) was higher in food secure group, all other variables showed higher measurements among the food insecure groups. Lipid profiles, TC (M=5.175, M=5.062) and LDL (M=3.100, M=2.914) were high for both male and female respectively, while TG is high for male (M=0.817) (p=0.037) and HDL for female (M=1.826). For body composition such as FMI (M=4.494, M=5.452), WC (M=77.46, M=76.82) and WHtR (M=0.471, M=0.497), male and female respectively, in food insecure group showed higher results but only FMI showed a significant difference (p = 0.016). Statistics showed an association between food security status and lipid profile (TG) and with FMI. However, no significant association was found with inflammatory marker. This study will continue further in depth in gene expression of peroxisome proliferator activated receptor gamma (PPAR-y) and endothelial dysfunction to better understand this issue. Regardless, current data provides knowledge and understanding of food insecurity experienced by young adults in university campus and may help them in making healthier food choices and be appreciative of the risk of chronic illnesses.

5.3 Introduction

Various research have been linked food insecurity with many adverse health outcomes including diabetes, hypertension and cardiovascular disease. The mechanisms by which food insecurity predisposes one to adverse health outcomes have not been well studied but are suggested to include shifts in dietary quantity and quality. It is thought that the food insecured individual will obtained foods that provide a higher caloric value per dollar spent; later, these high-calorie diets lead to obesity and direct to the associated adverse health outcome. Obesity that was thought one of the consequences of food insecurity is the one that was blamed for the adverse health outcome in those groups. However, this model does not account for alternative pathways whereby food insecurity may lead to poor health through malnutrition.

5.4 Brief Literature Review

Food insecurity is defined as "the limited or uncertain availability of nutritionally adequate, safe foods or the inability to acquire personally acceptable foods in socially acceptable ways"(1). In general definition a food insecure individual may have one or several of the following characteristics; 1) insufficient quantity of food; 2) limited diversity of food groups; 3) poor safety of food; and 4) procurement of food in socially unacceptable manners(1). Previous study in rural Malaysian population has shown that more than 50% of the household were experiencing some sort of food insecurity where obesity was strongly associated with that condition(2). Various research have been linked food insecurity with many adverse health outcomes including diabetes, hypertension and cardiovascular disease(3,4). The mechanisms by which food insecurity predisposes one to adverse health outcomes have not been well studied but are suggested to include shifts in dietary quantity and quality(5). It is thought that the food insecured individual will obtained foods that provide a higher caloric value per dollar spent; later, these high-calorie diets lead to obesity and direct to the associated adverse health outcome(5). Obesity that was thought one of the consequences of food insecurity is the one that was blamed for the adverse health outcome in those groups(5). However, this model does not account for alternative pathways whereby food insecurity may lead to poor health through malnutrition. In conjunction with that, it is possible that the stress and shifts in dietary patterns that characterize food insecurity stimulate an inflammatory state and alters immune function in food-insecure individuals that could start atherosclerosis formation. Specific nutritional markers, including vitamin A, vitamin B12, and folate, have been identified in biological and epidemiological studies as associated with inflammatory states and innate immune function(6,7). We also hypothesised that the food-insecure individual will also develop oxidative stress in their body. High calorie intake such as fat could further incite inflammatory response. It often begins with passive influx of LDL-c to the sub-endothelial space. In return endothelium of the blood vessel wall begins to express the adhesion molecule, VCAM-1(8). Once within the intima, the LDL-c will further be oxidized by reactive oxygen species (ROS) as a result of negative imbalance of body anti-oxidant defence mechanism such as catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx) and uric acid(9). Malondialdehyde (MDA) and F2-isoprostane would be detected as a result of lipid peroxidation in biological system(10). VCAM-1 activation attracts monocytes, which then migrate through the endothelial layer(8). Once within the arterial intima, the monocytes transform into the macrophages, engulf lipids, and become foam cells(8). T-lymphocytes will further activated and migrate into the intima and make the initial lesion of atherosclerosis worsen into fatty streak(8). Furthermore, complex plague will developed from the fatty streak as a result of progressive inflammation. Various cytokines also been released as a results of inflammatory response such as TNF-a and IL-6(11). This in return triggers liver to produce acute phase protein such as