

The Development of Diets to Induce Atherogenic Lipid Profiles for Cynomolgus Monkeys in Their Country of Origin

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Abstract Understanding the process of atherosclerosis progress can be studied in laboratory animals, such as nonhuman primate (NHP). Investigators at Bogor Agricultural University Indonesia, Primate Research Center (IPB) reported to develop an atherogenic diet (IPB 1) by using fresh egg yolk and coconut oil as source of cholesterol and fat. The aims of the research were to correct nutritional inadequacy in the initial IPB 1 atherogenic diet by supplementation with corn oil (IPB 1+CO); to use dry powdered egg yolk (PEY) instead of fresh egg yolk (IPB 1+CO+PEY); to use concentrated source of protein (43%) pupae meal (PM) instead of soya meal (IPB 1+CO+PM); and to use crystalline cholesterol (CC) instead of egg yolk (IPB 1+CO+CC). Twenty four *Macaca fascicularis* were used as animal model for three months adaptation followed by 12 months for four diet treatments. Parameters measured such body weight, waist circumference, trunk length, adiposity index, nutrient utilization, and plasma lipid profile every three months. This experiment used Completely Randomized Design with four treatments and six replications. Result showed that there were no significant differences found in morphometric parameters among the diet groups compared to one another or change from baseline. The nutrient (protein, fat and carbohydrate) consumption and the absorption were essentially the same for all four diet groups. The IPB 1+CO diet, the IPB 1+CO+PM diet and the IPB 1+CO+CC induced a similar atherogenic plasma lipid profile, with marked increases in total plasma cholesterol concentrations.

Keywords: atherogenic diet, crystalline cholesterol, powder egg yolk, pupa meal

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1. Introduction

Cynomolgus monkeys (*Macaca fascicularis*) have become the most comprehensively characterized nonhuman primate model of human atherosclerosis (cite or monkey chapter). They have been the model of choice for a wide variety of studies involving the effect of nutrition, social behavior, reproductive status, depression, obesity, hypertension and a number of other presumed risk factors for coronary artery atherosclerosis. Additionally, the cynomolgus monkey model has been used extensively to evaluate the effects of drugs, hormones and lifestyle issues such as exercise and social isolation on the progression and regression of atherosclerosis. It was reported that non human primates are phylogenetically closer to humans and have similar lipid metabolism as humans, meanwhile rodents have less different lipid metabolism from those humans [3].

Studies of the natural history of human atherosclerosis in Western societies have provided clear evidence that by young adulthood (35 years of age) the majority of subjects have developed fatty streaks and plaques [9]. Consequently, for a nonhuman primate model to be of the most translational value in the evaluation of interventions intended for the treatment or prevention of atherosclerosis of adult human beings, the model should have a comparable amount of atherosclerosis to that seen in adult human beings when the intervention to be evaluated is initiated. To accomplish pre-experimental induction of atherosclerosis comparable to that of human beings in Western societies requires about 12 to 16 months of feeding a diet that induces a significantly atherogenic lipid profile. That long delay in starting an intervention is a major handicap for investigators working in a research intensive environment. A potential solution to this long delay is the development of atherogenic diets prepared from ingredients available in the monkey's country of origin allowing monkeys to be fed the diet for an extended time at the supplier's facility before exporting them to the