seemed worthwhile to find out whether such a type of variation in nociceptive responsiveness followed a regular rhythm, using data that had been accumulated for the past three years and the results obtained provide the basis of this report.

Swiss OF_1 Strain, 4-5 weeks old (20-28 g), maintained at standard laboratory conditions were used throughout the study. Nociceptive reactions were measured using the hot plate technique previously described[3, 4] The apparatus consisted of a hot plate on which was placed a restraining cylinder (height 17 cm; diameter 13 cm). The temperature of the hot plate was 55° C. Animals which did not respond after 180 sec were removed from the hot plate "cut-off time". All these experiments were carried out between 2·00 p.m. and 6·00 p.m. The latencies are expressed as means ± S. E. M. (table I)

TABLE I

Annual variation in latencies to jump and lick (sec) ± S. E. M. in mice

Month	*Responses* Jumping latency	Licking latency	No of mice used (*n*)
January	90 ± 2	13 ± 1	150
February	94 ± 4	14 ± 1	60
March	96 ± 6	11 ± 1	20
April	84 ± 4	9 ± 1	40
May	76 ± 3	8 ± 1	80
June	66 ± 4	9 ± 1	70
July	64 ± 3	9 ± 1	50
August	64 ± 4	9 ± 1	20
September	84 ± 5	11 ± 2	20
October	108 ± 6	13 ± 1	30
November	118 ± 4	13 ± 1	30
December	98 ± 4	12 ± 1	80

The temperature of the hot plate was 55° C. "Cut-off time" was 180 sec.

Marked variations in nociceptive reactions were noticed. There was a gradual reduction in jumping latencies from March to August, after which they showed an increase. A similar trend was also observed in the licking latencies.

Our findings point to a possible role for endorphin systems in the annual regulation of nociception. Our data agree with that of Codd and Byrne[2] who reported seasonal fluctuation in the apparent number of [^{3}H] naloxone binding sites in mouse brain homogenates. These investigators also noticed that [^{3}H] naloxone binding was less in June, July and August, a period where we found that the latencies of nociceptive reactions were small. [^{3}H] Naloxone binding in the brain homogenates was greater from October through March and correspondingly we observed that the latencies to lick and jump were longer. The reduction in jumping latency produced by naloxone was smaller in June, July and August, whereas it produced greater hyperalgesia in other months (unpublished observations). Hence it is conceivable that oscillations in nociceptive sensitivity might be due to rhythmic fluctuations in the concentration of endorphins. This possibility remains to be confirmed. Whatever the underlying mechanisms might be, the present analysis clearly demonstrates an annual variation in the reactivity to thermoalgesic stimuli in mice.

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28 *September* 1981

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EFFECT OF AGE ON PLASMA LIPOPROTEIN CHOLESTEROL IN NORMAL SUBJECTS

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IN recent times, several clinical and epidemiological studies have emphasised the importance of plasma lipoprotein cholesterol and the development of coronary heart disease and atherosclerosis.[1-3] Many studies have been carried out in western countries to find out the normal pattern of lipoprotein cholesterol[4,5] which is significantly different from that of underdeveloped countries[5]. The age is the most important factor in the development of coronary heart disease and atherosclerosis. No report is available in literature regarding the normal levels of plasma lipoprotein cholesterol in healthy Indian subjects of different age groups. The present study therefore has been undertaken to find out the normal pattern of lipoprotein cholesterol in healthy individuals of different age groups in Chandigarh area.

Clinically healthy male individuals (numbering 382) of varying ages were considered for the present study and were grouped according to their age. Group I(87), Group II(93), Group III(95) and Group IV(107)

TABLE 1

Values of total and lipoprotein cholesterolin normal subjects with different age (Mean ± S.D)

Group	Total cholesterol (T-c)	HDL-c	LDL-c	VLDL-c	T-c / HDL-c	Blood pressure Systolic Diastolic
I(87)	201·7 ± 21·2	65·3 ± 10·2	92·3 ± 36·7	42·4 ± 14·2	3·2 ± 0·9	118 ± 8/82 ± 6
II(93)	231·5 ± 30·2[a]	59·1 ± 14·2[b]	123·2 ± 32·2[c]	60·7 ± 15·4[c]	3·94 ± 0·7[b]	125 ± 11/85 ± 8
III(95)	235·3 ± 36·7[b]	55·2 ± 13·2[c]	124·3 ± 48·7[c]	62·7 ± 24·8[c]	4·1 ± 0·7[c]	127 ± 10/89 ± 9
IV(107)	239·4 ± 40·2[b]	56·2 ± 14·2[c]	122·7 ± 39·7[c]	59·7 ± 19·7[c]	3·9 ± 0·7[b]	122 ± 10/89 ± 7

HDL-c High density lipoprotein cholesterol
LDL-c Low density lipoprotein cholesterol
VLDL-c Very low density lipoprotin cholesterol

a, $P<0·05$; *b*, 0·01; *c*, 0·001

aged 20–29 years, 30–39 years, 40–49 years and 50 years and above respectively. Fasting blood samples from healthy individuals were collected into EDTA vials (1 mg/ml of blood). Plasma was separated and processed for lipoprotein fractionation using heparin, manganese chloride and sodium dodecyl sulphate double precipitation techniques[6,7]. The cholesterol content in different fractions of lipoproteins was estimated[8].

In the present study, significant increase in the mean total cholesterol, low density lipoprotein cholesterol (LDL-c) and very low density lipoprotein cholesterol (VLDL-c) observed in the age group above 30 years (table 1). This may be because of decreased endogenous metabolism with the advancement of age. Significant increase in high density lipoprotein cholesterol (HDL-c) and decrease in LDL-c observed in Indian healthy individual as compared to age and sex matched healthy individuals of western countries[4,10] may be because of high intake of fat by western people. VLDL-c in Indian subjects was significantly higher as compared to those of the western countries in same age and sex matched groups.[11] This may be explained on the basis of the increase in carbohydrate intake with less of fat by Indians as compared with their counterpart in the west, resulting in an increased synthesis of VLDL.

10 *November* 1981

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DISTRIBUTION OF COMPLEMENT FIXING ANTIBODY TO EQUINE RHINOPNEUMONITIS VIRUS IN FOALS AND STALLIONS

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RESPIRATORY disease in young horses and subclinical infection in adult horses owing to equine rhinopneumonitis virus (ERV) have been recognized in many parts of the world[1–3]. Reports on ERV infection caused by equine herpes virus type 1 (EHV1) in India are generally restricted to abortion syndrome in mares[4,5]. Preliminary serological survey of ERV infection among army horses and mules in India