SEASONAL VARIATIONS IN BODY WEIGHT, SKINFOLD THICKNESS, FOOD INTAKE, SERUM LIPIDS AND ADIPOSE FAT COMPOSITION IN YOUNG MEN IN ANTARCTICA

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D.L. Easty

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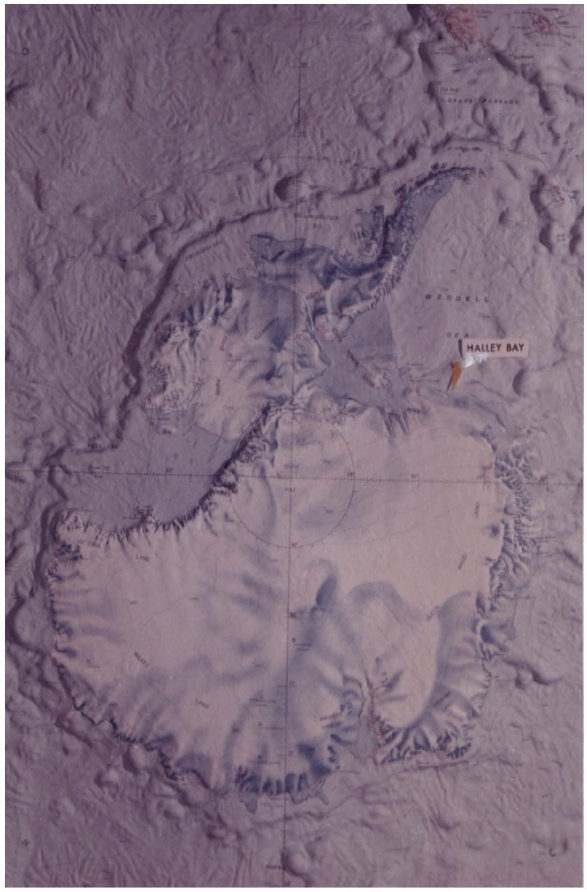

Plate i. A relief map demonstrating the position of Halley Bay on the Antarctic continent.

PREFACE

The present work was carried out while the author was acting as medical officer to the British Antarctic Survey Base, Halley Bay, situated on the coast of the Weddel Sea (75.36°S; 26.39°W, Plate i). The bases are maintained on this barren continent for the purpose of the exploration and the mapping of these areas comparatively new to mankind, and the recording of meteorological and geophysical data.

Each year, Halley Bay is visited by a relief ship which brings in supplies and new personnel. Except for the short period that the vessel is present, the base is entirely isolated from the outside world, and the only communication with civilisation is via radio contact with the Falkland Islands, close to Cape Horn.

The base consisted of a group of huts situated on a floating ice shelf, a continuation of the continental ice cap which constantly slides off the main land mass. The base lay 30 miles from the nearest point of the Antarctic coast, and moved approximately 500 yards each year towards the sea. The ice shelf, which was 600 feet in thickness and in places ranged up to 1000 feet, was characterised by its lack of scenic relief, being a featureless plain of snow which stretched far into the distance. On this ice, the living huts, generator sheds and laboratories of 25 men were placed. Though initially built on the surface of the ice shelf, the wooden huts were eventually covered by drift snow, so that hatches were constructed in order to enter the huts. The main living hut on the arrival of the 1961 Expedition lay 30 feet beneath the snow surface, and was in poor repair. During the first three months of the year (February to April 1961) the men strove to construct the outer shell of a new living accommodation, and at this time, the average expenditure of energy was at a maximum level for the year (Plate ii).

Halley Bay in the past was known as a static base, i.e. it

Plate ii. During the first 3 months of the year, the men constructed new living quarters. The illustration shows the sleeping quarters; other accommodation similar in size was joined by a short passage.

existed purely for purposes of geophysical research, but in 1961, husky dogs were introduced and a survey of the area was initiated. Thus the men of the base were able to take part in journeys which demanded considerable physical effort. The energy expenditure during sledging was far greater than the expenditure during normal base life.

It is often assumed that men taking part in Polar expeditions are frequently at a loss to know what to do with their time. This is an incorrect assumption, for to keep a base running smoothly and fully maintained demands the greater part of all men's time. Many of the men spent the full quota of their waking hours working up scientific results, or overhauling necessary machinery.

The Antarctic expedition member is subject to the following four main hazards:-

1) low temperature
2) isolation
3) the continuous darkness of the polar night
4) the risk of traumatic injury or fatal accident.

It is sufficient to point out that the first three hazards exert their main influence during the winter months; the low outside temperature and the darkness cause the men to spend almost all their time in the confines of the base huts. This is the period in which the sense of isolation is greatest and boredom due to lack of stimulation from the never changing environment can cause a degree of psychological tension. The risk of trauma, fatal or otherwise, is ever present, but is greatly reduced by a close attention to safety precautions by the expedition. Cold injury can and does occur to a minor degree but severe frostbite is unknown in members of the British Antarctic Survey.

Men on Antarctic expeditions are chosen because of their physical and mental fitness, and are usually between 20 to 30 years old. It is therefore the exception rather than the rule for the medical officer to be called upon to treat serious illness. This allows him to follow physiological research projects without impediment. These southern bases are well suited to two main types of experiment, namely -

1) those designed for the elucidation of the effects of cold on man, and
2) those which investigate variables which may not be influenced by low temperatures, but which may be affected by seasonal alterations in diet, energy expenditure, or psychological tension.

An Antarctic base, due to its isolation, coupled with the continuous availability of subjects could be regarded as a metabolic ward with interesting potentiality. It was with these fundamental issues in mind that the present work was initiated under the auspices of the Medical Research Council Division of Human Physiology in London. The project contains experiments which measure the effect of cold on the human, and also investigates parameters which are unlikely to be influenced by low temperatures.

It must be recognised that in a research project of this nature, in conditions which are often very different to those which would be found in a laboratory at home, the experiment is completely dependent upon the full cooperation of the members of the community. Thus, utter stringency in the control of certain variables cannot always be insisted upon due to the deleterious effect it could have on this cooperative effort.

Although the experiment was mainly concerned with the demonstration of seasonal changes and group differences, it is important to remember a third factor, the search for any change which might be indicative of a process of acclimatization to cold exposure. It can be

envisaged that a seasonal change would involve a rising and falling with the seasons, while a change due to acclimatization would demonstrate itself in two fundamental ways: a rise or fall to reach a constant level in the first few months; or a more prolonged trend lasting longer than a year, which would present as a continuous upwards or downwards slope during the year.

The purpose of the present investigation is:-

1) To determine whether any change occurs in the fatty-acid composition of the subcutaneous fat during exposure to cold.
2) a. To measure the dietary intake throughout one year as a comparative parameter to the serum lipid estimations, and to define the effects of environmental and seasonal changes, and to determine the effect of differing individual occupations within the social group upon the calorie intake.
   b. In conjunction with the dietary studies, to measure weight changes and variations in skinfold thickness, and to determine the relationship between these two variables.
3) To measure blood pressure and pulse rate, to determine the seasonal influence, and to compare individual readings with serum lipid levels.
4) To determine the effects of seasonal alterations in diet and physical activity upon serum lipid levels in a group of normal young men, and to ascertain the effects of a high fat intake with high energy expenditure, as exemplified during dog-sledging journeys, upon the lipid levels.
5) To demonstrate the individual variations in a single serum lipid level, and to examine each person's lipogram for abnormalities which, it has been suggested, may be of value in deciding the

risk of the later development of atheromatosis and/or ischaemic heart disease, and to demonstrate whether a subject showing such an abnormality also shows in other variables under examination, e.g. dietary intake, blood pressure, obesity, and body weight, changes said to be of significance in the pathogenesis of ischaemic heart disease.

It is convenient to consider the experiment as consisting of four separate parts, namely:-

SECTION III	The study of changes in subcutaneous fat;
SECTION IV	A diet survey, with weight and skinfold thickness estimates;
SECTION V	Changes in blood pressure and pulse rate;
SECTION VI	The serum lipid changes, with relevant observations where indicated made between the changes in dietary intake, body weight, and blood pressure and pulse rate.

SECTION I. BACKGROUND

A. THE INFLUENCE OF COLD ON THE PHYSIOLOGY OF THE HOMEOTHERM

1. Observations on Animals Adapted to Cold.
2. Observations on Laboratory Animals Subjected to Cold Stress.
3. Observations on Humans Indigenous in Cold Regions.
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B. DIET, SERUM LIPIDS AND CORONARY ARTERY DISEASE

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A. THE INFLUENCE OF COLD ON THE PHYSIOLOGY OF THE HOMEOTHERM

Man originated in an environment which complied with the physiological mechanisms of homeostasis of his body temperature. The occurrence of predators, or the quest for food may have influenced the construction of the first weapons and the move from the place of origin. Migration led to latitudes at which the tropical temperatures of man's natural environment no longer prevailed. Colder climates probably stimulated the wearing of clothing, and later, the revelation of fire. With the discovery of clothing, man became emancipated from his climatic environment, and the migration to yet higher latitudes was made possible. Yet can this be the complete picture? Is there physiological and anatomical evidence that man has adapted himself to live in regions where environmental temperatures are low, and are there grounds for supposing that the human when subjected to cold stress becomes acclimatized ?

From the outset, it is important to define the meaning of the terms adaptation, acclimatization, and acclimation. Hart (1950) suggested the following definitions:-

Acclimatization	Changes in the responses of the organism produced by continued alterations in the environment.
Acclimation	Alterations related to changes in a lifetime.
Adaptation	Changes occurring during a period of several generations.

1. Observations on Animals Adapted to Cold

It is convenient first to examine the generalised adaptive

changes which have occurred in mammals and birds in cold regions, and later to discuss the more localised changes. Irving (1957) made the startling observation that the range of temperatures to which Arctic animals are adapted varies between -50°C and +20°C, while the equivalent range for unclothed man lies between +23°C and +28°C. Arctic mammals and birds achieve thermal equilibrium in the lower portion of this range mainly by an increase of their insulative fur layer. Scholander, Walters, Hock and Irving (1950) in measuring the body insulation of some Arctic and tropical animals reported that the relationship between the fur thickness and its insulation in clo units, was approximately linear. These findings afford verification of the thickness law, which now clearly recognises the thermal insulation of clothing or fur is proportional to the thickness of dead air (Burton and Edholm, 1955).

Experiments have been performed which showed that the remarkable adaptation of Arctic homeotherms was largely dependent upon thermal insulation, by which the degree of cold to which they were adapted was greatly increased in comparison with the tropical animal. The work of Scholander, Hock, Walters and Irving (1950) on the critical temperatures of a series of mammals ranging from tropical to Arctic is interesting. The critical temperature can be defined as that environmental temperature which will cause the heat production of the organism to be raised by a 'chemical regulation'. Scholander et al indicated that in arctic mammals, the environmental temperature has to be extremely low before metabolism is increased. Thus the eskimo dog did not show an increment of its metabolic rate until the temperature had dropped to -30°C, while, on the other hand, the tropical racoon increased its metabolism when the temperature fell below 10°C. It is undeniable that group behavioural factors also play their part in

increasing the resistance of the organism to cold stress, as in the case of the seasonal migrations of arctic animals and birds, or the huddle formation of Emperor penguins (Prevost, 1962).

Adaptation to cold is shown by a more localised manifestation. It is interesting to speculate on the value of some gradients in the thermoregulatory control of body temperature. Scholander and Schevill (1955) have described the close proximity of the venae comitantes to the arterial supply in the tail fin of the whale. The artery has four to five veins deeply embedded in its wall. Hence the arterial supply is cooled by the returning venous blood, and the ultimate heat loss is greatly reduced. By this method of tissue cooling, the organism conserves body heat. It can also be postulated that peripheral vascular changes will act to increase heat loss, and therefore to reduce a body temperature which may be elevated.

Evidence of gradients in other mammals and birds also strongly suggests peripheral vascular adaptive changes. Irving and Krog (1955) reported large superficial temperature gradients in the limbs and the head of some arctic animals. In a dog exposed to an ambient temperature of $-30^{\circ}C$, the superficial temperature beneath the thick fur at the top of the thigh was $35^{\circ}C$, on the top of the pad and between the toes it was 8 to $16^{\circ}C$, and the temperature of the skin of the toe pad was $0^{\circ}C$. On the nose, similar gradients were found. At the same time, these changes were also shown to occur in the superficial tissues by the insertion of needle thermocouples. Without this peripheral cooling, it is clear that the heat loss would be greatly increased, especially in the case of the toe pads of mammals, which are in contact with the snow surface. For tissues to function at low temperatures some degree of adaptation is suggested at the cellular level from these findings.

In the majority of living things, the stored fat exists in the

liquid state. In the case of mammals, hard fat would be distinctly disadvantageous in that freedom of movement would be inhibited. There is a certain amount of evidence which suggests that ambient temperatures may influence the melting point, and hence the fatty acid composition and the iodine number of the fat stores of the organism. This is particularly true in the case of the stores which are situated close to the surface of the organism.

Irving, Schmidt-Nielsen and Abrahamson (1957) followed up the discovery of the characteristic cooling of the tissues of arctic mammals which, they pointed out, appeared to be an essential part of adaptation. They measured the melting points of marrow fat at intervals down the leg of the animal. In a reindeer, they found that the melting point fell from 50°C in the marrow fat of the femur, to 18°C in the terminal phalanges. However, Irving and his coworkers experienced some disappointment in their discovery that similar temperature gradients occurred in the marrow fat of the limbs of the panamanian brocket deer, which is a tropical animal.

Further to the above observations on the melting point of marrow fat Henriques and Hanson (1901) in studies on pigs demonstrated an inverse relationship between environmental temperature and the extent of depot fat unsaturation. In their measurements of the composition of the depot fats of the pig Dean and Hilditch (1933) showed that the least saturated fats occurred superficially, and that saturation increased with the depth below the surface at which the sample was taken. They concluded that the degree of unsaturation was inversely proportional to the local temperature at the site of the biopsy. In addition, Fischer, Hollands, and Weiss (1962) analysed samples of subcutaneous fat for fatty acids and iodine values in hens maintained at three environmental temperatures. It was found that fat from hens maintained at 0°C was

significantly more unsaturated and contained more di- and hexonoic acid than fat from birds kept at either 21°C or 32°C.

It is relevant to the foregoing that Pearson and Raper (1927) showed that on culturing the fungus aspergillus niger at various environmental temperatures, the amount of unsaturation of the composite fatty acids altered. As the temperature decreased, the degree of unsaturation of the fatty acids increased, with a resultant depression in melting point of the fat. Also Hilditch (1951) reported that in sunflower seeds the relative proportions of oleic and linoleic acid may vary considerably, and that such variations are conditioned mainly by the temperature of the locality from which the seeds were obtained. Hilditch stated that low temperatures tended to produce a more unsaturated mixture of fatty acids, with the converse for higher temperatures. The changes were confined to the unsaturated fatty acids, and this suggested a separate biochemical synthesis in the build-up of unsaturated acids.

2. Observations on Laboratory Animals Subjected to Cold Stress

Experiments were planned essentially to demonstrate the change in various parameters which might signify an increased tolerance and acclimatization to cold. Investigations concerning the value of various food items in aiding the resistance of the organism to cold stress have been performed in the hope that the conclusions drawn might ultimately be of benefit to man. Opinions are still varied as to the ideal composition for a diet in cold regions. Thus the food chosen by white rats in high and low ambient temperatures was observed by LeBlond, Dugal and Therien (1944). The rats were kept in environmental temperatures of -2°C, room temperature, and +32°C for 7 months, and were allowed the choice of several foods. In the cold the rats chose a diet, the

composition of which was 25% protein, 25% fat and 30% carbohydrate. The rats confined in the higher ambient temperatures consumed 70% of the total calories as carbohydrate, while the fat intake demonstrated a marked fall. Templeton and Ershoff (1949) in their work on survival times of rats which were fed single food items in the cold, showed that in acclimatized animals, those on a margarine or sucrose diet survived longer than rats fed on a casein or cotton seed oil diet. The rats which were fed casein or cotton seed oil survived no longer than starving rats.

Beaton (1963) has recently performed more detailed experiments on the survival of rats in the cold. He showed that rats fed on a 40% protein diet did not exhibit typical increases in total food consumption and in urine volume when exposed to cold, and further, that these animals appeared to be able to maintain body weight and to synthesise body fat during cold exposure. Beaton concluded that dietary protein levels may play an important role during exposure to cold.

The most important feature of experiments planned to ascertain the effect of a cold environment has been the measurement of the metabolic rate in laboratory animals, and in the greater number of cases this has been the rat. It has been shown that the metabolic rate increases when animals are exposed to low environmental temperatures and that this increase is dependent upon the critical temperature of the animal. Rubner (1902) designated this increase as being due to a chemical regulation, and furthermore went on to classify the mechanism whereby the animal controlled heat loss by sweating and peripheral circulatory changes as physical regulation. The argument as to whether the increase of metabolism in the cold is only due to a shivering response, or that it is to some extent due to another source of heat has continued for a long time. Consistent failure to demonstrate non-shivering thermogenesis led many to disbelieve in its existence.

The effect of cold acclimatization upon the animal's ability to maintain a constant internal temperature was investigated by Blair, Dimitroff and Hingeley (1951). This group found that when non-acclimatized rabbits were placed in a cold environment for 8 hours, their colonic temperatures fell by an average of $5^{o}F$, whereas a cold acclimatized rabbit showed a fall of only $0.1^{o}F$; they found similar changes in the colonic temperature of rats under the same conditions. They also reported that the cold acclimatized animals utilized twice as many calories as the unacclimatized, when subjected to cold stress. However, during a 5-hour exposure the weight loss was shown to be greater in acclimatized animals than in those which were unacclimatized. Blair et al concluded that the greater weight loss in the acclimatized animals indicated an increased metabolic utilization of body materials for heat production in order to maintain body temperature and to prevent cold injury.

In 1955, Davis and Mayer reported a series of experiments which demonstrated a dual mechanism of thermogenesis in rats. By means of a diathermy technique which increased the core temperature but did not affect the skin temperature, combined with curarisation, Davis and his group concluded that the heat production in the cold of unacclimatized animals consisted of two theoretical fractions; the basal fraction, upon which was superimposed a shivering fraction, which amounted to approximately 55% of the total cold induced metabolism, and a non-shivering fraction (due to central stimulation) contributing about 45% of the total cold induced metabolism in the rat. Davis (1958) went on to suggest that during the cold acclimatization of rats, oxygen consumption due to shivering thermogenesis is progressively reduced to reach zero by the 15th day, while non-shivering oxygen consumption due to peripheral stimulation rises from 0 to a maximum in which it supplies 50% of the basal level of rats in a normal environment. He suggested

that cold induced metabolism at any point in time during the period of acclimatization is the algebraic sum of shivering, of skin-stimulated non-shivering heat production, and of centrally stimulated non-shivering heat production.

Further investigation of the effect of cold upon the laboratory animal led to a search for changes in endocrine function. Much decisive data which had accumulated before the work of Davis et al had shown that the hypothesis for a chemical thermogenesis could not easily be dismissed, and this was partially based on the evidence of changes in the visceral organs.

Sellers and You (1950) measured the metabolic rate of two groups of rats after they had been subjected to a cold stress. The first group were measured at 1.5°C and the second at 30°C. While shivering occurred in the first group there was none in the second. They found that the metabolic rate at 30°C as well as that at 1.5°C rose as the time of the previous cold exposure increased. Further they showed that the metabolic rate at 30°C of cold acclimatized and control rats fell by about 70% when they were anaesthetised, but the cold acclimatized rats still had a higher metabolic rate than the controls. These results pointed to a rise of metabolism in non-muscular tissue.

Sellers and You then showed that the increment in the metabolic rate at 30°C in cold acclimatized animals was associated with an increase in thyroid weight and activity, the latter being assessed from the appearance of the thyroid and from the increase in thyrotropic activity of the anterior pituitary.

Sellers, You and Thomas (1951) demonstrated the essential role of cortical and thyroid hormones in prolonging survival in the cold. Adrenalectomised or thyroidectomised cold acclimatized rats survived in a temperature of 1.5°C for 12 days, while similarly treated unacclimatized

rats died in less than a quarter of this time. The longer survival time of the cold acclimatized rats, the authors suggested, was due to the residual levels of the hormones circulating in blood. Furthermore, they pointed out that if the period between operation and cold exposure was lengthened to 2 weeks the survival time of rats was reduced to a few hours. These experiments clearly demonstrated the importance of the endocrine balance of the organism in the ability to resist cold stress.

In the latter portion of the last decade, further facts accumulated to support this hypothesis for cold induced non-shivering thermogenesis. You and Sellers (1951) showed an increased oxygen consumption with surviving liver from cold acclimatized rats as compared with in vitro studies of oxygen uptake of liver slices from unacclimatized rats. Others have confirmed and extended these findings; notably Weiss (1954) to kidney slices as well as to skeletal muscle, and Hannon (1958) showed that homogenates of liver from cold acclimatized animals showed acceleration of oxygen uptake. Greater significance can now be attached to these in vitro findings by virtue of direct thermal measurements of organs in vivo, which showed that during cold exposure the metabolism demonstrates a cyclic pattern, during which the liver temperature rises ahead of temperature increases in the muscle bed (Donhoffer, Farkas, HangLaszlo, Jarai and Szegvari, 1959).

The invitro and in vivo experiments are interesting in the light of some extensive data collected some years ago by Emery, Emery and Schwabe (1940), concerning the exposure of rats. The rats were subjected for 16 hours to a temperature of 2^{0}C, and for the remaining 8 hours they were removed to the control temperature of 26^{o}C. This data has been recomputed by Brody (1945) on the bases of the relationship between the various organs of the rat and the body size in order to correct for the changes in the latter during the period of exposure,

and was quoted in an extensive review on the cellular responses in cold acclimatization written by Smith and Hoijer (1962). The figures indicated that on the 15th day of cold stress, weight gains were achieved in all the metabolically important visceral organs including the lung and the adrenal and thyroid glands. From the subsequent measurements on the 30th and 60th days, it was evident that further gains were registered in the thyroid and kidney, but not in the heart nor in the liver, which if anything showed some regression during the last two readings. Thus from these results it appeared that the heat production originally supplied by the liver at the 15th day was taken over by all other visceral organs with the possible exception of the heart.

In the preceding paragraphs, some of the evidence for both adaptation and acclimatization have been described. Work on the critical temperatures of arctic mammals and birds has indicated that the insulation of the organism is of prime importance to the survival of the organism under conditions of cold stress. Metabolic studies made on laboratory animals demonstrated the undoubted importance of shivering thermogenesis and of non-shivering, or chemical thermogenesis, in maintaining an increased metabolic rate under cold stress. However, researches did not cease at this point and a mass of evidence has been gathered concerning the more detailed mechanisms of temperature control. In vertebrates the recognition of neural, neurohumeral, endocrine and motor functions capable of meeting the cold stress with compensatory thermogenesis opened up investigations at the cellular level. It is unnecessary to report this later work in great detail. It is sufficient to point out that the laboratory animal has been found to adjust to a cold environment in the main by the elevation of metabolic rate. In

the acute phase of the homeothermic adjustment shivering plays some part, but later tends to give way to the gradual development of a non-shivering thermogenesis. This non-shivering heat production represents, metabolically, a new steady state of thermal adjustment marked by the enlargement of some visceral and endocrine organs relative to total carcass mass, an increased total metabolism, and by a rise in the oxygen consumption of isolated liver, heart, kidney and muscle. The status of homeothermy at reduced ambient temperatures is thus maintained by resort to greater heat production, although alternative solutions have also been achieved in the field through behavioural and insulative adjustments.

3. Observations on Humans Indigenous in Cold Regions

One of the ways in which the Eskimos were thought to increase their resistance to cold was by their unusual diet. The question of whether their high protein intake was of benefit in their resistance to cold, or whether it was merely a case of supply controlling demand has been frequently investigated.

The typical dietary pattern of the Eskimos has been described by Sinclair (1953) in some detail. He pointed out that during the winter months the Eskimos' food consumption consists mostly of seal. In the summer when the Eskimos migrate inland, their food intake is supplemented by caribou and wolf flesh, salmon, hare, duck and geese. They also cease to be strict carnivores and eat berries and roots. Much of the meat is eaten raw. Sinclair recomputed the data collected by Rink (1877) of the annual food consumption of 6100 Eskimos in the Southern Inspectorate of Greenland. He estimated the total per capita food intake to be worth 3359 kcal/day and that 47% of these calories were supplied by protein, 46% by fat and only 7% by carbohydrate. The

percentage composition showed close agreement with the data of Krogh and Krogh (1913) but did not correspond with the result of Bertelson. Bertelson (1935 to 1943) showed that in Greenland Eskimos the total calories in winter were composed of 20.3% protein, 51.3% carbohydrate, and 28.4% fat; in the summer fish eating season the composition was 41.6, 48.3 and 9.9% respectively. However, in this survey about 65% of the food was imported. Assessments of food intake of Eskimos by Rabinowitch and Smith (1936), Hoygaard (1941) and Brown, Bird, Boag, Delahaye, Green, Hatcher and Page (1954) all showed similarity in the percentage of calories supplied by the three food groups, the salient feature of which was the high percentage supplied by protein and fat. Only in the results of Bertelson did the percentage of calories supplied by carbohydrate show an increase.

In a recent survey made by Mann, Scott, Hursh, Heller, Youmans, Consolazio, Bridgeforth, Russel, and Silverman (1962) food consumption was measured in two different Eskimo groups: firstly in Eskimo guardsmen who were in training, and who were physically active, and secondly in a village community of civilian Eskimos. The average calorie intake of the first group was 4665 kcal/man/day and that of the villagers was 1855 kcal/day. The food available for the National Guardsmen was unlimited, whereas the village community were not so privileged. The percentage of calories supplied by protein, fat and carbohydrate was for the guardsmen respectively 15%, 37% and 48%, while that for the villagers was 30%, 36% and 34%. Thus the guardsmen on their unlimited diet largely used carbohydrate to increase their caloric intake.

Hoygaard (1941) discusses the importance of a high fat diet, and cites subjective evidence both from the indigenous population and from other reports that Eskimos cannot maintain "warmth" on a diet composed largely of lean meat. Eskimos are aware that they must have a

high proportion of their dietary intake supplied by fat. On the evidence it appears that the high protein intakes in the past, and to a lesser extent during the present day, merely resulted from food availability, and are of no greater value than a western diet in aiding the ability to resist cold. However, in the light of the very recent work of Beaton (1963) which demonstrated that cold exposed rats could function more efficiently on a high protein diet, the tendency for Eskimos to lower their protein intake without ill effect is interesting and cannot be adequately explained.

The measurement of the basal metabolic rate in the primitive Eskimos has frequently been carried out, and has given rise to some controversy. Three hundred and forty basal metabolic rate estimations were made on 73 Eskimos from four different locations in Alaska by Rodahl (1952). When the increments in metabolism accountable to apprehension and protein metabolism were eliminated, Rodahl concluded that the metabolism was exactly the same as in white control groups.

Høygaard (1941) showed that the B.M.R. was 13% over the Dubois standard in 20 Eskimos and this result was close to the assessment of Rodahl. Hoygaard also mentioned the dependence of the raised metabolic rate on the protein metabolism. Two of the subjects used in his survey subsisted upon a 'western' diet, and demonstrated only slight elevation. The mean result reported by Heinbecker (1928) of +33% is however too high to be interpreted as being due to protein metabolism, and Hoygaard suggests that this very high value may have been due to experimental error.

Levine (1940) found that the basal rates of Eskimos at Point Barrow, Alaska, were similar to white people living in the same area. The greater proportion of the evidence is in favour of a raised metabolic rate, although it is unlikely that this is an adaptive change, but is

dependent on the extraordinarily high protein intake of the race.

Eskimos show evidence of local adaptation to cold, which can be compared with the peripheral cooling of the extremities of arctic mammals. Coffey (1954) showed that in manual dexterity tests in a cold room the loss of efficiency by the Eskimo and Indian groups was far less than that of a group of white controls. Coffey reported further investigations which measured sensory motor functions, which are generally accepted as being components in the decline of manual dexterity. Thus this worker found with Mackworth's V-test, a two point discrimination measurement, combined with a strength of grip test and a joint flexibility test as developed by Hunter (1952), that the native population was far superior to the white control group. The decline under cold conditions in the Eskimos and Indians was half as much as the decline in the white controls. Taking the findings of Brown, Boag, Bird, Delahaye, Green, Hatcher and Page (1954) that the Eskimo has a smaller fall of hand blood flow on exposure than white subjects, Coffey postulated that his findings to some extent suggested a local tissue acclimatization. In Brown et al's series of experiments, they showed that the temperature gradients in muscle and subcutaneous fat were greater in Eskimos than in whites, and yet these workers were also impressed by the high degree of manual dexterity which remained in the hand of the Eskimos.

Bazett, Love, Newton, Eisenberg, Day and Forster (1948) have shown that a venous arterial heat exchange occurs in the vascular supply and drainage of the hand, which would account for the low forearm temperatures in Eskimos. This is similar to the heat exchange which occurs in the vascular architecture of the tail fin of the whale observed by Scholander and Schevill (1955).

Frequently, workers have been forced back into the position of accepting that though animal experiments have demonstrated well marked

changes due to acclimatization, the Eskimo is not subjected to the same order of cold stress as would exist in laboratory experiments, and hence does not show such changes. The Eskimo has achieved emancipation from the thermal demands in the same way as the arctic mammal, by the efficient insulation of his body surface. Studies on mankind in the cold regions will doubtless continue to yield unfruitful results unless a group exposed to a higher degree of cold stress can be discovered. Two ethnological groups, however, are subjected to severe cold on occasions, and in the following, the experiments carried out on them will be described.

Wyndham and Morrison (1958) have investigated the adjustment to cold of the Bushmen of the Kalahari Desert. This nomadic group has failed to show intellectual development and in all temperatures the tribesmen wear a loin cloth and a leather cloak. Night temperatures are liable to fall to around $10^{o}C$, and the heat loss of the sleeping Bushman is further enhanced by radiation loss to the sky. However, Wyndham et al in this comparative study of Bushmen and whites could ascertain no evidence of an acclimatization to the lowish nocturnal temperature from observations made on skin temperature, core temperature and shivering. They concluded that the Bushman has, in his own primitive manner, produced a microclimate during the coldest part of the day which is near the thermoneutral zone; furthermore, that his adaptation is an intellectual and not a physiological one, in that he has developed thermal barriers by means of primitive clothing, fire and rudimentary shelter.

The impressive experiment of Scholander, Hammel, Hart, LeMessurier and Stein (1958) on a tribe of central Australian aborigines did however yield some evidence of possible physiological adaptation to cold. The criterion of adaptation used by Scholander et al was that the subjects sleep deeply without showing evidence of cold stress in the

form of shivering. The metabolic rate, with skin and core temperatures, were measured in both whites and aborigines sleeping unclothed in light sleeping bags, the insulative value of which was 1.9 clo. The aborigines were observed to sleep well, while the whites experienced a sleepless or disturbed night with frequent shivering. The depressions of skin and core temperatures were similar in the two groups; the metabolic rate in the whites was often fluctuant and raised, whereas in the natives the metabolic rate was non fluctuant and was below the basal level.

In normal circumstances the native uses a fire and windbreak to overcome cold stress in the manner of the Bushmen of the Kalahari, but at the same time the aborigine seems to be able to tolerate cold to a greater extent than the white. The metabolic rate was lower than basal, and hence there was no suggestion of a chemical regulation. It may be that the Australian aborigine can tolerate peripheral cooling as an economical response, resembling the cooling of the extremities in arctic animals and birds.

Whether man, normally domiciled in temperate regions, shows physiological changes on being subjected to a cold environment has been investigated extensively, as evidenced by the great quantity of literature produced in the last two decades. The ability of the human to survive in polar regions has hinged on two lines of investigation; firstly, one based upon experiments over short periods of time in which variables which might influence the acclimatization and tolerance to cold have been assessed, and secondly, an empirical one which has evolved in the field over many years of polar exploration.

4. Observations on the Short Term Effects of Cold on Man

Measurements have been made of the basal metabolic rates in

temperate and cold conditions, and also the cost of standard work under varying ambient temperatures.

Horvath, Freedman and Golden (1947) demonstrated a 30% increase in oxygen consumption in subjects while at rest in a cold room, and showed that a slight increment of oxygen consumption, compared to the pre-exposure level, remained when the men were returned to normal room temperatures. Horvath and Golden (1947) measured the energy expenditure of standard work under temperate and cold conditions, and found a 10% elevation in expenditure in the reduced temperatures which appeared to be unrelated to the amount of clothing worn, but Eliot, Stein and Bader (1948) in investigating cross-acclimatization to heat and cold found no differences in metabolic rates in hot and cold environments. Gray, Consolazio and Kark (1951) in estimating the energy expenditures of men in cold, temperate and hot environments reported that:-

a) the calorie output for a given amount of external work performed at a constant temperature increased about 5% when clothing was changed from desert to temperate, and increased by 5% more when clothing was changed from temperate to arctic;
b) the energy expenditure for a given amount of external work performed in a given outfit of clothes decreased about 2% as the temperature was raised from -15°to +60°F and decreased by a further 2% when the temperature was raised 60°F to 90°F.

In measuring the diurnal pattern of oxygen uptake before, during, and after cold exposure, Iampietro, Bass and Buskirk (1957) found a substantial rise in oxygen consumption during the periods of cold exposure, but the 'before' and the 'after' exposure assessments were much the same.

The evidence for acclimatization in man seems incontrovertible from the experiments reported by Davis (1961) and Davis and Johnston (1961).

Davis established that as men are artificially acclimatized they show a fall off of shivering, but that heat production does not change. The groups were subjected to a standard cold stress at regular intervals. Furthermore, Davis and Johnston demonstrated that male clerical workers showed a seasonal change in their response to standard cold stress; in the summer, the metabolism and shivering were high, whereas in the winter months the shivering fell off to a greater extent than the heat production. This evidence was strongly in favour of a non-shivering thermogenesis in the human. It showed agreement with the animal studies of Davis et al performed in 1958 in which during acclimatization, shivering thermogenesis fell off, while the non-shivering thermogenesis was progressively increased to reach a plateau level.

An experiment that provided some interesting and positive results was initiated by Scholander, Hammel, Anderson and Løyning (1958) who measured skin and core temperatures with metabolic rates in groups of unacclimatized and acclimatized students. The acclimatized group had existed for 6 weeks in one of the cold regions of Norway, wearing light clothing and sleeping in a bag which provided less than 1 clo insulation. As in the case of the work of Scholander et al (1958) on Australian aborigines, the measurements were made on the sleeping subjects. The experiments indicated that foot and shoulder skin temperatures in the lightly clad sleepers feel to a greater extent in the controls than in the acclimatized individuals. The findings contrast with those of the aborigines in which the decrements in skin temperature were similar to the white control group. However, the fact that the acclimatized Norwegians slept more deeply than the controls, who thrashed about continuously throughout the night, shows agreement with the observations reported for the aboriginal subjects. In the experiments performed in Norway, the acclimatized men showed a slight tendency to raise their

resting metabolic rate when sleeping, and this also occurred when they were exposed to an ambient temperature of 20°C in the nude. Hence Scholander et al concluded that white acclimatized subjects showed increased expenditure of metabolic energy when sleeping, at the same time keeping the temperature of their feet at a comfortable level. The aborigines however merely showed a greater tolerance to low temperatures, demonstrating a sub-basal metabolic rate with similar skin temperature decrements as those of the white subjects.

Recently Heberling and Adams (1961) have cast some doubt on the fairness of attributing the changes in skin temperature to cold acclimatization. They reported that after physical training the surface temperatures of the extremities remained elevated on exposure to cold, as compared with measurements before the period of training. A difference in physical fitness between native populations and the European controls might well account for the changes found between the two groups of Scholander et al.

Studies of the nutritional intake of men subjected to cold have indicated that either a high fat or a high carbohydrate diet can increase resistance to cold. One of the most often quoted studies pertaining to food intake at differing ambient temperatures is that of Johnson and Kark (1947). These workers measured the dietary intake of army personnel following standard activities in tropical, temperate and arctic regions. The mean total calorie intakes were respectively 3300, 3850 and 4400 kcal/man/day. Johnson and Kark reported that there was little variation in the percentage composition of the total calories in the three climatic extremes; they concluded that the increased calorie intake shown in the cooler climates resulted from the hobbling effect of external clothing.

A project reported anonymously (1946) indicated that although

-the average individual developed more resistance to cold on a high fat diet, the tests also showed that performance ability was improved by a high carbohydrate diet. Dugal, LeBlond and Therien (1945) had previously shown that diets rich in fat were superior to those rich in carbohydrates in developing resistance to cold. However, Keeton, Lambert, Glickman, Mitchell, Last and Fahnstock (1946) reported that a high carbohydrate consumption was superior to a high protein intake in increasing man's tolerance to intense cold. It is likely therefore that the high protein intakes of Eskimos result from the availability of foods, and not from choice.

5. Observations on Men on Polar Expeditions

Perusal of the literature devoted to physiological research on members of expeditions to cold regions is disappointing in the light of the greater number of positive findings in animal experiments, and to a lesser extent is overshadowed by the results obtained from human cold chamber experiments. Many observations have been made by medical officers returning from cold regions. Bingham (1948), Liversidge (1949) and Butson (1950) each reported increased intake and tolerance of fat in cold regions. Simpson (1959) reported an eosinopaenia in subjects after manhauling sledge journeys, and after periods of mental stress; there was no indication that the changes were particularly the result of cold.

Several workers have noted a seasonal change in body weights in individuals living at polar bases (MacLean, 1919; Kalnenas, 1951; and Wilson, 1960) in which the usual pattern is a gain in the winter months, followed by a loss in the summer months. Lewis, Masterton and Rosenbaum (1960) demonstrated a correlation between weight changes and skinfold thickness changes. Detailed measurements have also been made of the skinfold thickness in some recent Antarctic expeditions by

Graham (1959) and Wyatt (1963), who have shown that the skinfold thickness correlates closely with the changes in body weight.

Whether the sharp increase in body weight and skinfold thikness which often occurs at the beginning of the year in expedition members in cold regions is a change due to acclimatization cannot be said; however, Baker and Daniels (1956) demonstrated that obese men maintained high rectal temperatures and lower skin temperatures when compared with thin men, in low environmental temperatures. But, further to this, both Goldsmith (1959) and Tikhomirov (1963) reported a loss of body weight during the winter months. However, Goldsmith made his measurements upon the advance party of the Commonwealth TransAntarctic Expedition, who were subjected to considerable cold exposure, and also their food was rationed.

One of the most significant findings in connection with the acclimatizational status of temperate man domiciled in polar regions was made by Massey (1959), who substantiated the cold chamber experiments of Mackworth (1953), in that a two-point discrimination test suggested that there was an increase of tactile discrimination when hands were subjected to a standard cold stress, with the length of stay in the cold. Sparke (1963) has since repeated these experiments and reported similar conclusions. Goldsmith (1960) produced convincing evidence of acclimatization to cold in Antarctica by keeping clothing records over one year. He showed that there was evidence of reduction of the number of layers of clothing worn in the second half of the year.

The long term investigation to determine the influence of the seasons in North Greenland on the metabolic rates in expedition personnel made by Lewis, Masterton and Rosenbaum (1961) indicated that basal levels showed no significant change over the year, and were in no way elevated above the normal. Similar observations were made by Lockhart (1941),

Butson (1949) and Wilson (1956). However, Milan, Elsner and Rodahl (1961) demonstrated a significant decrease in metabolic response to a standard cold stress in men who had lived for 3 months in the Antarctic and suggested that the changes represented a physiological adaptation to chronic cold.

Budd (1962) has recently reported an experiment carried out at Mawson, situated on the Antarctic continental land mass. Budd found significant changes in the serial measurements of the core temperature of four subjects. Control experiments performed in Australia before and after the expedition showed that when the men were subjected to a standard cold stress, the rectal temperature fell. However, when the subjects were exposed to the standard cold stress in Antarctica, Budd showed that a significant rise occurred in the core temperature. The greatest response occurred in the spring when the environmental temperatures were at their minimum. This field experiment indicated that a physiological change may occur which increases man's resistance to cold.

The comparative lack of positive information produced from physiological data recorded in Antarctic expeditions' personnel may be explainable by:-

a) the difficulty in making physiological observations in conditions which are often adverse and unstimulating, and
b) by the fact that subjects on these expeditions are thermally isolated from the environment, and that the true exposure is frequently small.

Norman (1960) demonstrated that scientists at Halley Bay spent 9% of their elapsed day out-of-doors, while the remaining 91% was passed in the warmth of the base hut. He also demonstrated that in winter activity was greatly reduced compared with that in summer.

The conclusions to be made from the first section of this introduction are, therefore, that though animal and human cold chamber researches have frequently suggested that there is evidence of cold acclimatization, little has been found in temperate man living for periods in cold areas. This has been well summarised by Edholm (1960) and Lewis and Masterton (1963).

Macpherson (1950) has stated that a temperate climate does not represent the neutral condition for man, i.e. one in which the environmental stress is minimum, and from which adaptation is possible both to hotter and to colder climates. He points out that man in a temperate climate is approaching the extreme range of adaptation to cooler conditions - that is maximum adaptation to cold. Macpherson eruditely concludes that it is fruitless to attempt to demonstrate profound physiological changes in temperate man on exposure to severe cold, the greater part of the possible adaptation in this direction having already been made, and that any further changes must necessarily be small.

Yet there is a lack of data concerning the dietary intakes of men in the field, and a long term survey designed to measure the seasonal influence on food intake has never been made. It would naturally appear that during the cold winter months, the food consumption would increase, but the fact that energy expenditure in the winter is reduced (Norman, 1960) could cast doubt on such an assumption. In addition, though there is quantity of data connected with body weight and skinfold thickness changes, it has not been elucidated to what the increases are due, whether to the laying up of protein stores, or to water retention, or simply to the increase in adiposity of the men. The whole picture of the causes of the weight changes in expedition personnel has yet to be clarified, and the experiment which is reported herein has attempted to provide some of the answers to the problem.

Evidence has been cited for the changes in the composition and melting point of adipose fat under the influence of cold in the pig and the hen. No data has been found by the author for the human which substantiates the changes found in laboratory animals. It is clear that, though the cold stress is small, a longitudinal survey of the fatty acid composition of the subcutaneous fat of temperate man in Antarctica should be carried out so that this index of acclimatization can finally be proved of either positive or negative value.

B. DIET, SERUM LIPIDS AND CORONARY ARTERY DISEASE.

1. Morbidity and Mortality Studies

The rising incidence of both mortality and morbidity from ischaemic heart disease since the turn of the century has caused concern and intensive investigation. The widespread optimism that the problem of atherosclerosis is not insoluble, is signified by the enormous volume of literature that has accumulated during the last decade.

Coronary disease, causing obstruction to the blood supply to the heart itself, is one of the major scourges of civilization. Coronary and other arteriosclerotic heart disease caused 17% of all deaths in England and Wales in 1961 (McMichael, 1963). In Britain alone, more than 100,000 people die from the disease every year.

Atherosclerosis is not a new disease. Ruffer (1911) has examined Egyptian mummies dating to 1500 B.C. and concluded that atherosclerosis was present in this population and Sandison (1962) has recently substantiated these findings by microscopic techniques. A review by Smith (1960) points out that this disease was discussed by Aristotle and his contemporaries (cc 400 B.C.) and that it has been touched on by many medical writers since. The fatty nature of the atherosclerotic lesion was emphasised a century ago by Vogel (1847)

who identified cholesterol as the major constituent of the atheromatous plaque, and by Virchow (1867), who described the progression of the fatty degenerative changes in the aorta.

Studies of the incidence of ischaemic heart disease have been made

a) as a function of time in the same population, and

b) by comparisons between varying population groups.

Clearly, Method a) has the advantage of eliminating factors due to heredity. However, in a population with a low incidence of ischaemic heart disease which migrates to an area in which the environmental factors alter, e.g. the Jews in Israel, the Japanese in Hawaii and the Negroes in Chicago, and in which the incidence of heart disease also changes, a genetic factor can largely be dismissed.

Statistical analysis of mortality rates in the Northern European countries for the years preceding, during, and after World War II showed that in the years of the war, deaths from circulatory diseases fell in Norway, Sweden and Finland (Biörck, 1956). However, Denmark showed no such trend. The war years were associated with considerable changes in nutritional intake, physical activity and mental stress; Biörck points out that the depression in mortality from circulatory diseases in Finland, Norway, and Sweden was reflected in a lowering of fat consumption during the same years. That Denmark, and for purposes of comparison, the United States, showed no depression in either mortality rates or fat intake, suggested to Biörck that the depressions in the Northern European countries in fat consumption and mortality rates might be related. However, the evidence that physical activity plays a role in the aetiology of ischaemic heart disease (Morris, Heady, Raffle, Roberts and Parks, 1953) is strongly positive, and it is probable that in general the population showed an increase in activity during the

war years. It can by no means be held that the variations in mortality in cardiovascular disease during the years 1935 and 1949 were absolutely attributable to the parallel alterations in the intake of dietary fat.

Differences in the incidence of ischaemic heart disease or atheromatosis between various ethnological groups provided renewed impetus in the search for causative agents in the pathogenesis of cardiovascular disease. Kimura (1956) reported that the death rate ascribed to coronary heart disease in Japan was approximately one-tenth of that in the United States. In a thorough survey of autopsy material, Kimura reported further that the incidence of advanced coronary sclerosis was again one-tenth of that in persons of the same age in the United States. Higginson (1956) confirmed the belief that the severer complications of atherosclerosis, coronary and cerebral thrombosis, angina pectoris, and intermittent claudication are rare amongst the native peoples of Africa. Post-mortem studies showed that the coronary vessels of hospitalised Bantu were far less atherosclerotic than those of Danish or American populations. Keys (1956) reported the low mortality from degenerative heart disease in Italians compared with Americans.

Evidence that differing mortality rates from ischaemic heart disease are related to geographical or climatic conditions was investigated; Larson (1957) reported a raised incidence of severe atherosclerosis in Japanese who had emigrated to Hawaii and California, and Brunner and Löbl (1957) recorded similar differences between early and recent immigrant Jews in Israel. However, the study of the mortality rates in the three racial groups of Cape Town showed that they were low for the Bantu and rose through the Cape Coloureds to reach a maximum in the European population (Bronte-Stewart, 1958). Additional evidence that the climatic influence may be ignored as a

disease agent is provided by the occurrence in the United Kingdom of differing mortality rates from ischaemic heart disease in various social classes (Logan, 1952) and by the occupational distributions of coronary deaths (Stewart, 1950).

2. Large Scale Serum Lipid Surveys

In epidemiological studies in races widely divergent in traditional custom and mode of life, and in the same race subjected to different environmental conditions, susceptibility to severe atherosclerosis and ischaemic heart disease appeared to be determined largely by environmental factors; of the many studied, differences in dietary fat intake appeared to be the most likely. In 1952, Keys (1956) initiated a series of epidemiological surveys which stemmed from the following four presumptions:-

i) That serum cholesterol level is related to atherogenesis. It is not the only factor involved, but its measurement should have predictive value for the tendency to develop atherogenesis and coronary heart disease in populations, though it is emphasised that it has very limited value in regard to individuals.

ii) Aside from metabolic diseases, the only factor that clearly may influence the serum cholesterol concentration is the habitual diet. In man, the cholesterol in natural diets has little or no effect but the total dietary fat is important.

iii) Experiments on animals cannot provide the desired information for man on the quantitative relationships between these variables. Controlled dietary experiments are difficult and costly, and they cannot be made equivalent to the experiments of nature extending over many years.

iv) Comparative studies of suitably selected different populations can test theories about the importance of the diet and other items

such as the mode of life and should provide practical clues to the prevention of atherosclerosis and coronary heart disease.

By 1956 Keys (1956) was able to show that in areas where the percentage of calories consumed as fat was above 40%, severe atherosclerosis was common and the serum cholesterol rose sharply with age. In groups eating little fat such as the South African Bantu (Bronte-Stewart, Keys and Brock, 1955), the Chimbu of New Guinea (De Wolfe and Whyte, 1958) and the Japanese (Keys, Kimura, Kusukawa, Bronte-Stewart, Larsen and Keys, 1958) severe atherosclerosis was rare and the rise of serum cholesterol with age was non-significant. Further Bersohn and Wayburne (1955) reported that at birth Bantu and European serum cholesterol levels were the same, that differences appear in early life and by the age of 20 years the significant differences are seen not only between races but between income classes within each race, so that it is unlikely that these differing age trends are explicable on genetic grounds only.

3. The Influence of Dietary Fat on Serum Lipids

That all fats did not behave in a similar fashion had been suggested by studies on vegetarians (Hardinge and Stare, 1954). Though this was denied from some quarters, controlled feeding experiments by several different groups (Kinsell, Michaels and Foreman, 1955; Beveridge, Connell and Mayer, 1956; Bronte-Stewart, Antonis, Eales and Brock, 1956; Ahrens, Hirsch, Insull, Tsaltas, Blomstrand and Peterson, 1957; Malmros and Wigand, 1957) all confirmed each other's findings. Essentially, these groups showed that animal fats such as butter, beef dripping or tallow, beef muscle fat, lard and eggs led to a prompt rise in serum cholesterol levels, whereas vegetable oils such as those from corn, sunflower, safflower, peanut and olive lowered them.

The vegetable oils are characterised among other things by their fluid nature at normal room temperatures, and a greatly increased content of mono- and polyunsaturated fatty acids over those of animal fats.

Bronte-Stewart (1958) pointed out that the difference in the effects of animal and hydrogenated vegetable fats on the one hand and natural vegetable and marine oils on the other hand must lie in the constituent fatty acids or a factor at present inseparable from them. Ahrens (1957) stated that if most of the non-fatty acid materials in dietary fats are removed either chemically or by molecular distillation, the characteristic response of the patient to that dietary fat is not lost. Ahrens further reported that, if the percentage differences between control cholesterol levels and those produced by dietary fats were calculated, these differences showed a linear relationship to the iodine value of the various fats which were tested. Removal of 80% of the non-saponified materials (such as sitosterols, carotenes and tocopherols) from corn oil failed to abolish its cholesterol lowering properties.

In the latter years of the 1950's two schools of thought emerged concerning the determining factor in the action of vegetable oils upon serum cholesterol and phospholipid concentrations. Kinsell and Sinclair (1957) postulated that the major factor in the highly unsaturated oils is their content of linolenic acid, and that hypercholesterolaemia and atherosclerosis are expressions of a deficiency of essential fatty acids (i.e. linolenic and arachidonic acids) in man. The views of Kinsell and Sinclair were weakened by the finding that on feeding oils with low content of essential fatty acids significant depressions of serum phospholipids, cholesterol and beta-lipoprotein were still manifest (Malmros and Wigand, 1957). Ahrens and his group

therefore conclude that it is the degree of unsaturation of constituent fatty acids of a dietary fat which produce the depression of phospholipid and cholesterol levels in serum.

Without entering upon the more complex mechanism in which the fatty acid composition of dietary fat influences serum lipid concentrations, the view expressed by Ahrens is now generally accepted. There can be little doubt that the variation of serum phospholipid and cholesterol levels in different population groups is largely dependent upon the chemical structure of the dietary fat.

4. The Triple Association between Diet, Serum Lipids and Atherosclerosis

Certain criticisms have been levelled at the conclusion that there is a triangular relationship between diet, serum cholesterol and atherosclerosis and ischaemic heart disease. The evidence for a direct association between diet and serum cholesterol trends is strong. Far less convincing is the strength of the other links in the triple association. That serum cholesterol levels have a value in the prediction of future coronary artery disease is an often made assumption which is not always found to be true and this weakens the argument for the triangular relationship. Keys (1957) has stated that any blood measurement could only indicate the atherogenic potential of the blood at the time at which the blood was drawn. Autopsies made during the Korean War (Enos, Holmes and Beyer, 1953) showed that young American soldiers had well developed atherogenic lesions by the third decade. The clinical appearance of the disease of a Western population in the 50's represents the cumulative effect of many years. Kagan, Dawber, Kannel and Revotskie (1962) in reporting some of the findings of the Framlingham study, a prospective study of coronary heart disease initiated in 1949, state that the total serum cholesterol is a satisfactory

index of coronary heart disease risk, at least in surveys of large populations.

The alarming increases in mortality relating to cardiovascular disease in Britain since World War II parallels the increase in the consumption of animal fat. Yudkin (1957) has pointed out the errors of attributing a correlation between the rise of animal fat intake and heart disease and stressed that statistically positive correlations do not imply a causal relationship. He demonstrated that many other parameters which have increased during the last two decades (e.g. number of television sets and telephones, and the intake of dietary protein) have a similar bearing on ischaemic heart disease as the dietary intake of fat. Bronte-Stewart (1958) has claimed that the rise in intake of animal fat was accompanied by a decrease in the consumption of unsaturated fatty acids. He has also suggested that the differences in occupational mortality could be related to the fact that the amount of dairy products and other animal fats eaten constitutes one of the most important social class differences in the dietary habits of man. Yet, further to this, it is hard to explain the mortalities shown to exist by Morris, Heady, Raffle, Roberts and Parks (1953) in bus conductors and bus drivers. The inactive bus drivers suffered approximately twice as many occlusions at the same age as did the active bus conductors. Keys (1957) has questioned the truth of the conclusion that activity is related to coronary thrombosis and points out that as activity is increased, the consumption of carbohydrates rises at the expense of dietary fat intake. However, it does seem inescapable that activity plays a part as the causative agent in atheromatosis and ischaemic heart disease, and experiments have been devised to demonstrate the influence of physical activity upon serum cholesterol (Keys, Anderson and Mickelson, 1956) and also upon postprandial levels of serum free fatty acids and triglyceride

concentrations (Nikkila and Konttinen, 1962); in both experiments physical activity showed a significant serum lipid depressing effect. It can be fairly stated that the investigation of the influence of physical activity upon the incidence of coronary artery disease demands further research to define its aetiological responsibility in the pathogenesis.

5. Pathogenesis of Atheroma

Concurrently with the evolution of the dietary fat-cholesterol hypothesis, research has been channelled into the pathogenesis of atherosclerosis. More than 100 years ago Rokitansky propounded the thrombogenic view of atheroma, namely that the lesions may be the result of organisation of mural thrombi clinging to the arterial walls (Hartcroft, 1960). Anitschow (1933) demonstrated that rats fed on a high-cholesterol diet developed atheroma.

The essential components of the human atherosclerotic plaque appear to be intimal fibrosis and lipid infiltration or degeneration. The relative amounts of fibrotic tissue and fatty deposits determines the hardness or softness of the plaque. Investigations have therefore centred around fat, fibrin, or both, but considerable controversy exists as to which component is primary and which is secondary. The time honoured approach in the study of the pathogenesis of any illness begins with the study of the sequential relationships of the component part of the lesions and is followed by an elementary theory of the fundamental mechanism of the pathogenesis.

Numerous studies of the pulmonary and systemic arteries in man have indicated that organisation of occlusive or mural fibrin thrombi will lead to fibrous intimal thickening and from these studies the thrombotic mechanism of atherogenesis has been suggested. Studies of the pulmonary

arterial lesions in patients with 'primary' pulmonary arteriosclerosis (Owen, Thomas, Castleman and Bland, 1953) mitral stenosis (Thomas, Lee, Rabin, O'Neal, 1956) and congenital heart disease (O'Neal and Thomas, 1955) have all helped to establish that these intimal lesions are in fact organised thrombi, their presence being closely associated with evidence of thrombo-embolism such as recognizable recent and organising thrombi or pulmonary infarcts and sources for thrombo-emboli in systemic veins and the chambers of the right heart. O'Neal and Still (1962) have described a theoretical sequence of events by which an occlusive pulmonary thrombo-embolus is transformed into a fibrous intimal plaque. The possibility whether, and the mechanism whereby lipids enter the thrombi, and the effect of lipids on the persistance and progress of the lesions, have not as yet been elucidated.

The filtration theory of Page (1954) was originally introduced by Virchow a century ago. The theory implies that lipid infiltration is the primary process and that the fibrous tissue present indicates a reaction to the presence of fat. The theory postulates that the centrifugal force of the circulation drives fat into contact with the arterial intima. Over the years, lipid deposition accumulates, and this is intensified by the presence of blood lipid in excess amount or in abnormal form, if the intraluminar or lateral filtration pressure is higher or if the endothelium of the vessel walls is abnormally permeable. Thus from the theory there is a satisfactory explanation for the particular localization of the plaques at sites of pressure or velocity change within the circulatory system, and for increased severity of atherosclerosis in the presence of hypertension and such disorders of lipid metabolism as diabetes mellitus and essential xanthomatosis.

O'Neal and Still (1962) performed experiments which

substantiated the filtration theory. Using the rat in which cholesterol-induced atherosclerosis bears a close resemblance to the early fatty streaks of man, they demonstrated the penetration of the circulating cholesterol containing foam cells into the subendothelial space to form the fatty streak. They suggested that a similar mechanism for the formation of the fatty streak may apply to man.

The possibility that the solubility of fats may effect the pathogenesis of atheroma, focussed attention on the lipoprotein complex. Being insoluble in plasma or body fluids, fats are combined with cholesterol, phospholipids and proteins in the form of lipoproteins, which are readily soluble. Three hypotheses have now been grouped under the heading of the colloidal macromolecular theories; in these theories, the emphasis is placed not so much on the quantities of serum cholesterol as in its physico-chemical status, i.e. the size of its micellar aggregates, the density and size of macromolecular complexes, and the stability of these colloidally placed particles in the plasma. The theories are:-

i) the macromolecular instability theory (Hueper, 1945)
ii) the lipoprotein theory (Goffman, Jones, Lindgren, Lyon, Elliott and Strisower, 1950)
iii) the chylomicron theory (Moreton, 1947)

The lipoprotein and the chylomicron theories are essentially modifications of the macromolecular theory, substituting specific concept of lipoproteins and chylomicrons for the more general concept of colloidally unstable cholesterol micels. The unstable complexes tend to leave the axial stream and congregate in the marginal portion. They then have close contact with the intimal lining and are disturbed more intensely by irregularities in the contour of the vascular lumen. Thus the instability of the colloidal complexes increases, and their aggregation and

precipitation on the intimal lining is favoured. If the precipitated particles are sufficiently large, they are not taken up by the lining cells, but provoke the formation of granulomas of the foreign body type. If the particles are of small size, they are phagocytized by endothelial and connective tissue cells, resulting in the formation of foam cells and reactive focal endothelial proliferations.

The series of events leads to the formation of atheromatous 'cushions', which may undergo secondary degenerative changes through the breakdown of foam cells and mononuclear cells, a process which results in the release of lipoidal material from the cells. Then there is the formation of cholesterol crystals, hyalinization, and calcification with finally the invasion of fibroblasts.

A third theory based upon intimal haemorrhages has been suggested by Paterson (1936). However, with the latter theory, and that of a thrombogenic mechanism, it is difficult to explain the presence of fat in the plaque. The extent to which this occurs does not encourage acceptance of the view that fat present is the product of blood destruction from small haemorrhages occurring over long periods. A filtration theory would explain the appearance of the fatty streaks.

Astrup (1956) has suggested that atheroma represents a disturbance in the balance of fibrin deposition and fibrin removal, and recently a great deal of interest has been shown in fibrinolysis as a possible pathogenic mechanism. In vitro studies reported by Bronte-Stewart (1960) demonstrated a great affinity of fibrin for cholesterol and certain of its esters (acetate, stearate and oleate). Little affinity appeared to exist between fibrin and triglyceride and phospholipids. Furthermore, cholesterol oleate inhibits fibrinolysis (Mitchell and Briers, 1959). It is possible that lipid enters the

atheromatous plaque at the time of fibrin formation and a large amount of lipid within the plaque may represent its earliest phase.

The evidence for a filtration theory is perhaps a little more convincing than for other hypotheses. And yet myocardial infarction cannot be explained by a triple hypothesis alone and many factors must each play a significant role. The work of Hartcroft and Thomas (1957) on the production of infarcts by dietary means in rats parallels the situation in the human. The rats which developed infarcts did not differ in their cholesterol levels from those which produced no myocardial infarction.

6. The Relationship of Atherosclerosis to Ischaemic Heart Disease

It has been generally accepted that atherosclerosis forms the basis of occlusive vascular disease of the heart, but its relationship to myocardial infarction yet remains to be clarified. Morris (1951) produced evidence that, though the incidence of coronary heart disease has increased, irrespective of an ageing population and improved diagnosis, the incidence of advanced atheroma may be decreasing. Morris pointed out that advanced atheromatosis occurs in autopsies from patients who have not died from myocardial infarction, while conversely, in spite of careful dissection, evidence of coronary occlusion in cases who have died from infarction often cannot be found.

Ischaemic heart disease was defined (World Health Organisation, 1957) as a cardiac disability, acute or chronic, arising from reduction or arrest of blood supply to the myocardium in association with disease processes in the coronary arterial system. It is evident that it will depend upon the following three interacting elements (Morris, 1951):-

i) atheroma, with narrowing of the coronary arteries, vasculisation of the intima, necrosis and haemorrhage;
ii) recent and old occlusion of the arteries;
iii) the collateral circulation.

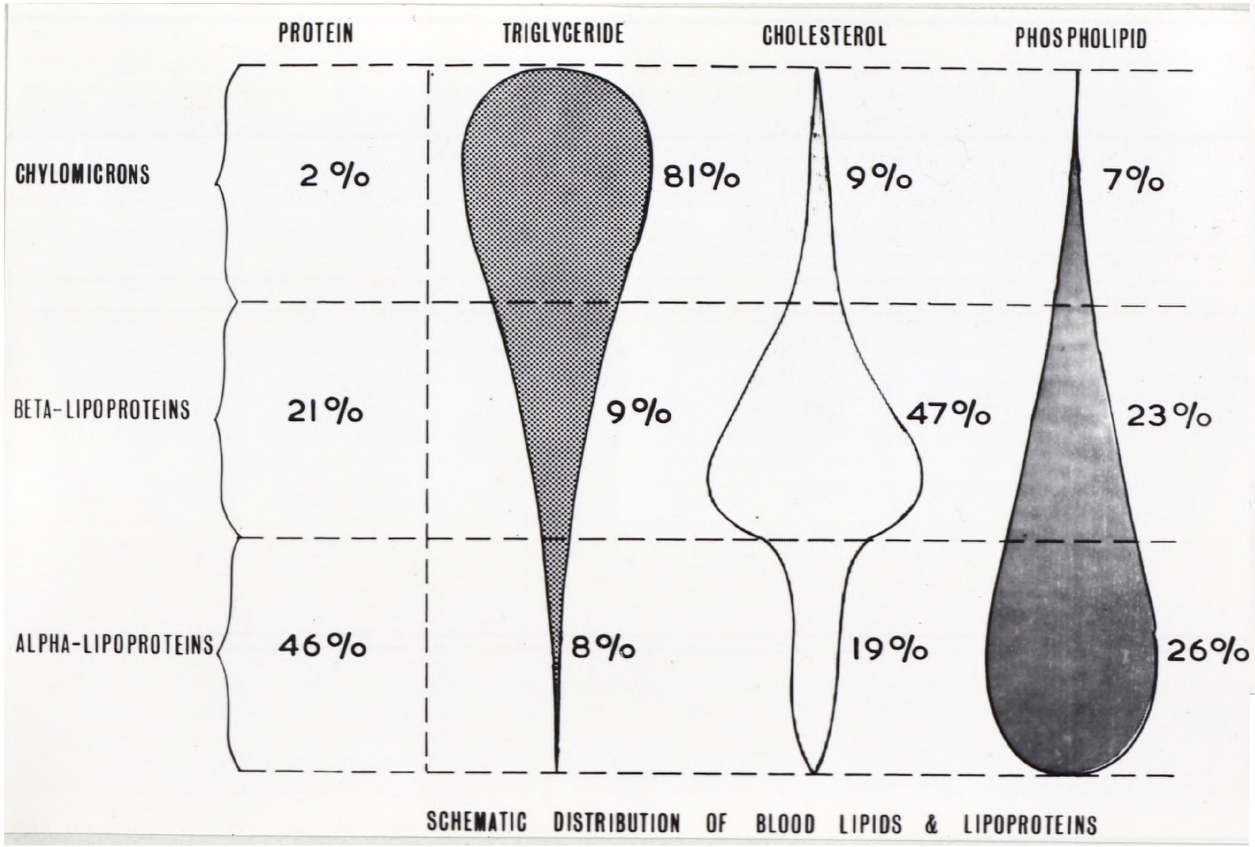
That narrowing of the coronary vessels by atheromatous plaque formation plays a role in the pathogenesis of ischaemic disease is undeniable, but it is conceivable that in addition to atherosclerosis a process may exist for the full explanation of the clinical manifestations. In recent years, there has been a revived interest in thrombotic and other mechanisms that interfere with circulatory flow. In ischaemic heart disease, the end product of atherosclerosis, multiple causes should therefore be considered; independent pathogenic mechanisms leading towards the final product may have varying status in separate population groups and therefore may be overemphasized in their responsibilities.

7. The Lipoproteins

Several recent articles have reviewed fat transport in man, and are concerned with the relationship of the lipoprotein system to coronary artery disease (Frederickson, 1957; Olson and Vester, 1960; Allbrink, 1962). Much of the research on lipoproteins has been directed towards the definition of suitable indices to enable one to predict the future development of ischaemic heart disease. In studies of homogeneous groups, particularly in connection with clinical practice, there is a considerable overlap between patients and controls (Bronte-Stewart, 1959).

The lipoprotein complex has been isolated and characterised both by electrophoretic and ultracentrifuge techniques (Lingren, Goffman and Elliott, 1951). Electrophoresis separates the lipoproteins in

Figure 1. A highly theoretical diagram demonstrating the compositions of 3 lipoprotein classes in human serum. (Taken from Frederickson, 1957; and Bragdon, Havel and Boyle, 1956).



three broad classes, the "alpha$_1$" and the "alpha$_2$" and the beta lipoprotein. Goffman classified the lipoproteins into two main groups, the low and the high density lipoproteins, based on ultrafuge techniques. The low density lipoproteins, which include the beta, and probably the "alpha$_2$" lipoproteins (Frederickson, 1957) can be further separated into a spectrum of classes lying within certain Svedberg flotation unit values which ascend numerically with increasing rate of flotation in the centrifugal field.

Bragdon, Havel and Boyle (1956) estimated the approximate percentage composition of four lipoprotein fractions:-

i) chylomicrons
ii) very low density lipoproteins (Sf > 10)
iii) low density lipoproteins (Sf 0-10)
iv) high density lipoproteins in normal subjects.

Bragdon, Havel and Boyle's figures (see Figure 1) indicated that the percentage of protein progressively rose from 2% in the chylomicron fraction to 46% in the high density (alpha$_1$) fraction, and conversely that triglyceride fell from 81% in the chylomicron fraction through 52% in the very low density lipoproteins to 8% in the high density fractions. The percentage of cholesterol in the low, and the very low density lipoproteins was greater than the percentage of phospholipid, while in the high density lipoproteins the proportion of phospholipid was increased over cholesterol.

It has been shown that age, masculinity and ischaemic heart disease favour a higher proportion of cholesterol in the beta lipoprotein fraction (Barr, 1953; Nikkila, 1953). Though phospholipids rise similarly to cholesterol concentrations, they do so to a lesser extent and the cholesterol:phospholipid ratio which is usually lower

than one, becomes greater than one (Gertler and White, 1954).

A large amount of data has been collected showing that it is the fasting beta-lipoprotein, or the low density lipoprotein which is abnormally high in many cases of coronary heart disease. Since the proportion of cholesterol in the beta-lipoprotein is greater than the proportion of phospholipid, it is evident that an elevation of the low density fractions will alter the normal ratio of cholesterol:phospholipid; furthermore, an elevation of the neutral fat or triglyceride would be expected, and proof which substantiates this has been obtained by Allbrink and Man (1959) who showed that patients with ischaemic heart disease have raised triglyceride levels. These workers believe that the measurement of triglyceride concentrations is a more satisfactory index of the likelihood of developing ischaemic heart disease than cholesterol levels. They point out that the overlap of cholesterol levels between coronary patients and controls is greater than the overlap using triglyceride estimations.

It is convenient at this point to enumerate the expected serum levels in normal subjects, and to compare them with the concentrations which have been found to occur in patients with iscaemic heart disease. Bronte-Stewart (1959) has reported the distribution of cholesterol in the alpha and beta-lipoprotein fractions in normal young men, and in patients with ischaemic heart disease. For young American males with serum total cholesterol levels of 197 mg %, the expected proportions of cholesterol in the alpha and beta-lipoprotein fractions would be 25 and 75% respectively. In patients with ischaemic heart disease, the levels have been shown to be 14% for cholesterol in the alpha-fraction and 86% for cholesterol in the beta-lipoprotein fraction.

Table 1 is taken from a paper by Oliver and Boyd (1957) and

shows the approximate ranges of the circulating lipids and lipoproteins in coronary artery disease.

Table 1. Ranges of serum lipids in normal subjects and patients with coronary artery disease.

	Coronary artery disease	Normal
Total cholesterol	200-300 mg %	120-240 mg %
C:P ratio	0.85 - 1.10	0.70 - 0.90
% cholesterol on beta-lipoproteins	80-100	60-85

8. Seasonal Changes in Serum Lipids

One way to demonstrate the influence of the environmental factors is the longtitudinal measurement of the beta-lipoproteins under conditions, for example, of changing physical activity or emtional stress. Such a situation would exist at an Antarctic continental base, in which the seasonal changes have considerable bearing on the dietary intake, activity and psychological tension. That psychological stress can cause elevation of serum cholesterol levels has been well shown by Friedman, Rosenman and Carroll (1958).

The work of Keys, Karvonen and Fidanza (1958) on Eastern Finlanders demonstrated a winter increase of 100 mg % in serum cholesterol levels and it appeared possible that similar changes might occur in men in an Antarctic base. Such a group would be subjected to similar environmental influences as the Finlanders of Keys et al. The authors made no decisive comment as to the reason for the seasonal change, except to state that it was due to environmental factors. By the measurement of these environmental factors, it was thought that a

prime agent for such a seasonal effect might be defined.

In addition to the seasonal changes found by Keys et al (1958) some earlier researches yielded some positive results. Currie (1924) for instance, who studied both normal subjects and cancer patients at Glasgow, found that the cholesterol values were highest in summer. Similar observations were made by Pucher, Griffith, Brownell, Klein and Carmer (1934) at Buffalo, and Villaverde and Vidal (1939) in Cuba. Pucher et al drew attention to the fact that they had noted a transient fall in cholesterol in the spring. In Rochester, New York, Kaiser and Gray (1934) observed that during the cold season the serum lipid values were higher than during the warm summer in both healthy children and children suffering from various diseases. They stated, however, that seasonal variations are suggestive though not conclusive.

In contrast to the above mentioned studies, in which seasonal variations in the serum lipid values were observed certain writers have failed to detect periodical changes accompanying the succession of the seasons. McEachern and Gilmour (1932) who performed their studies in Winnipeg observed no seasonal variations in serum cholesterol. Man and Gildea (1937) of New Haven noted no such variations in the values for serum cholesterol, phosphatides and fatty acids, and Turner and Steiner (1939) also failed to notice seasonal variations in the cholesterol levels on hospital patients in New York. The Swedes Josephson (1947) and Josephson and Dahlberg (1952) determined the cholesterol levels of certain subjects in the spring and in the autumn without detecting any significant differences. In Finland, Konttinen (1959) found that the mean values for serum cholesterol were the same in groups of young men called up for compulsory military service in February, June and October, respectively. This finding he regarded as evidence against the occurrence of seasonal variations.

In 1961, Thomas, Holljes and Eisenberg published a study of seasonal variations in the serum cholesterol levels of 25 convicts (aged 22 - 28 years). In Baltimore, they determined the serum cholesterol once a month from December 1958 to November 1959, noting clear seasonal variations, the values being highest in the winter months and lowest in the late spring, summer and early autumn. In a group of 16 convicts in whom determinations were made every month the mean values for cholesterol were 260-265 mg % in December, January and the following November against 214.5 mg % in May and 216.5 mg % in June.

It is of interest that Jervell (1960) found myocardial infarction to be more common in winter than in summer. In 1013 cases of fatal coronary thromboses in Hamburg, Dotzauer and Naeve (1956) found a similar trend. However, in an investigation of 193 cases of myocardial infarction, Lindholm (1963) found no seasonal variation in frequency of morbidity or mortality due to the disease. The possibility exists therefore that the seasonal changes which are known to occur in mortality from ischaemic heart disease may result from concurrent changes in serum lipid concentrations.

The effect of cold environments upon serum lipid levels has frequently been examined in animals but not in man. Recently Hannon and Durrer (1963) have reported seasonal variations in blood lipid concentrations in husky dogs with a marked winter elevation. However, it would be unlikely for cold induced lipaemia to occur in the human while in Antarctica; Norman (1960) as well as demonstrating the low degree of outside exposure, showed that the sublothing temperature rarely dropped below the critical temperature of man.

The beta-lipoproteins may be defended as the agent of athero-

sclerosis on the basis of Koch's principles (Olson, 1959). On the other hand Olson and Vester (1960) have pointed out that in man on an abundant diet, they may be less important as a cause than other factors such as hypertension, tortuosity of vessels and hereditary predisposition. Thus they summarise by denoting the beta-lipoprotein as interacting with, on the one hand, the environmental factors of diet, drugs, exercise, occupation, culture, and stress, and on the other hand factors intrinsic in the host, namely age, liver function, arterial reactivity, haemodynamic status, endocrine status and psychic status. That dietary fat intake plays a role in the aetiology of atheroma is indisputable, and yet to accept that as the complete picture, and to act upon it is a questionable move (Masters and Jaffe, 1963). To attempt to change the diets of a whole civilization on evidence thus far obtained is an extreme measure which would have far-reaching economic consequences; but to treat either patients with ischaemic heart disease, or those in whom the blood lipid levels are markedly elevated and who are known to develop coronary artery disease to a greater extent than controls (e.g. familial hypercholesterolaemia, diabetes mellitus, hypothyroidism) is indicated.

It was concluded in the first part of this introduction that the changes due to acclimatization to cold in men taking part in polar expeditions appear to be minimal, or non-existent. Yet variations in activity, body weight and skinfold thickness have been well documented. Groups of men in isolation have the advantage that many variables can be examined in great detail. The human is a comparatively rare laboratory animal, and circumstances in which he may be studied for long periods of time are even more scarce.

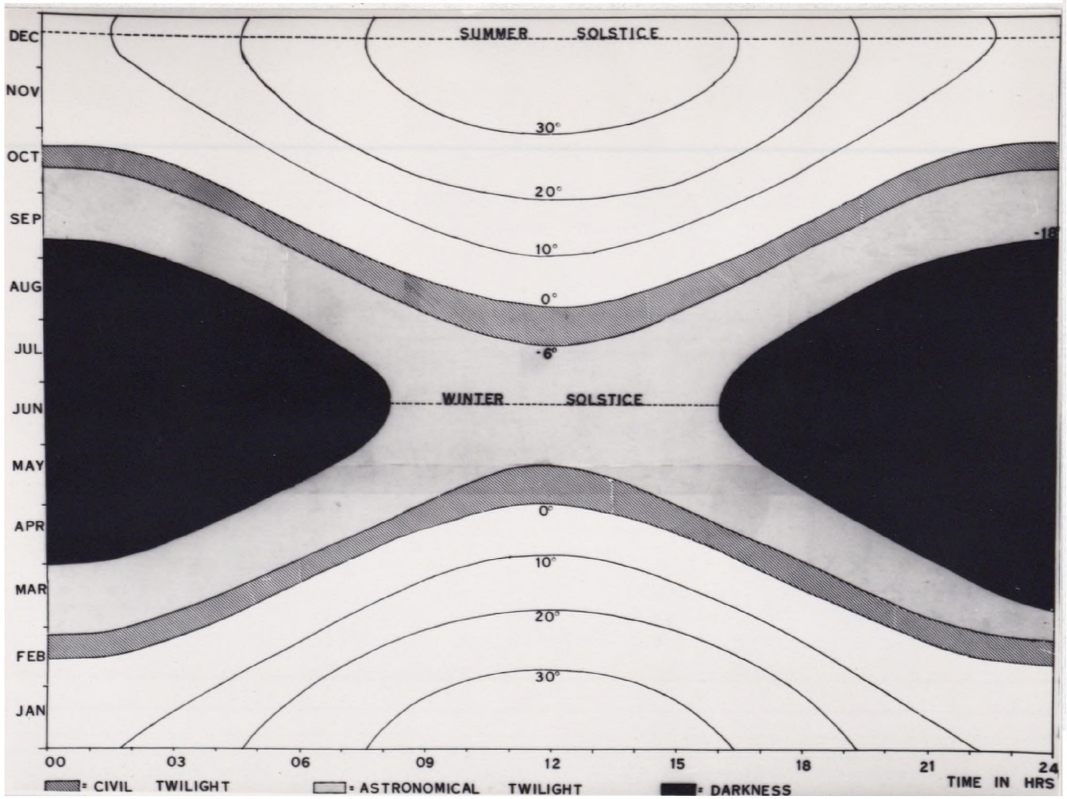
The current theories concerning the relationship between

dietary fat, serum lipids and ischaemic heart disease have many unanswered problems, and it was considered that a survey of these lipids in young active males over a year in the Antarctic would provide additions to present knowledge.

SECTION II. THE ENVIRONMENT AND THE MEN

1. The environment.
2. The men.
3. The diets.

Figure 2. Isopleth-diagram of solar altitude during one year. Drawn for Halley Bay (Lat. S 75° 0.7').



The experiment was designed to measure the individual levels of certain parameters over a period of one year and the effect of environmental factors on these parameters. Statistically, this would be termed as a search for a 'man effect' and a search for a 'monthly' or a 'seasonal effect'. In addition, the comparison of the values gained in Antarctica with estimations made in similar groups of subjects in the United Kingdom, or more broadly, in temperate climates would perhaps allow conclusions to be made. The two main external variables in the study are the environment and the men.

1. The environment

Two environmental factors act together to produce three seasons in the year at Halley Bay. Figure 2 demonstrates the isopleth diagram of solar altitude for the latitude on which Halley Bay was situated. Civil twilight is defined as the time when the true position of the centre of the sun is 6° below the horizon, at which time darkness forces the suspension of normal outside activity. Astronomical twilight is defined as the time when the position of the sun is 18° below the horizon, and there is no trace of twilight glow. The isopleth diagram shows that from the beginning of May until the first week in August, i.e. a period of approximately three months, the darkness is absolute and incapacitating. From the third week in October to the second week in February, a period of approximately four months, the sun does not set. During the remaining five months it is seen that there are variable combinations of light darkness during the 24 hours.

Figure 3 and Table 2 show the monthly means of the outdoor temperatures from February 1961 to January 1962 at Halley Bay. The

Figure 3. The monthly mean outside temperatures (central line) with the mean values of the daily extremes, for Halley Bay. The horizontal interrupted line demonstrates the mean annual temperature.

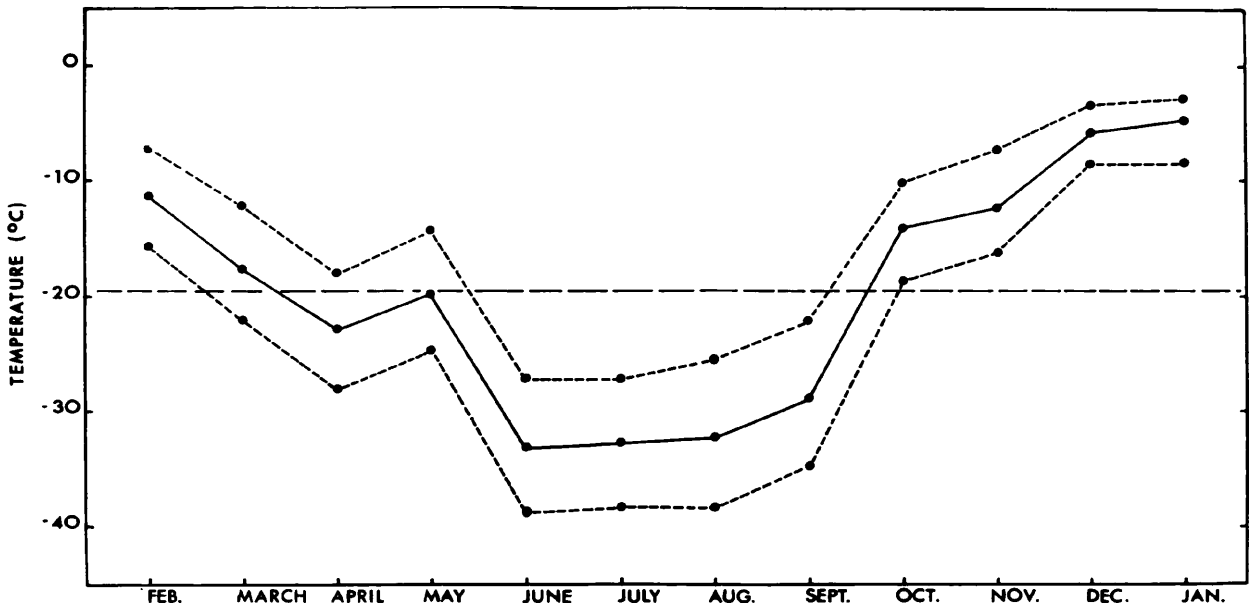


Table 2. Meteorological data for Halley Bay

	Feb	Mar	Apr	May	Jne	Jly	Aug	Sept	Nov	Oct	Dec	Jan
Monthly means Temperature (°C)	-11.3	-17.6	-22.9	-19.7	-33.1	-32.7	-32.3	-28.8	-14.2	-12.4	- 5.8	-4.9
Mean monthly maximum temperature of daily extremes (°C)	-07.1	-12.1	-18.0	-14.2	-27.1	-27.2	-25.5	-22.2	-10.3	-07.3	-03.5	-02.9
Mean monthly minimum temperature of daily extremes (°C)	-15.7	-22.0	-28.0	-24.6	-38.7	-38.4	-38.4	-34.8	-18.6	-16.2	-08.5	-08.5
Wind Speed monthly mean (knots)	10	14	12	18	11	8	10	9	21	10	12	8

maximum temperature at no time rose above 0°C and it was a comparatively rare occurrence for temperatures to be recorded higher than zero. In January 1962, a measurement of 3°C was recorded, and this was the highest temperature reported from the base since it was established in 1957. The meteorological data indicates that the monthly average wind speed did not show a seasonal variation, whereas the outside temperatures fell considerably during the winter period.

For the purpose of the experiment, therefore, the year could be divided into three seasons, viz.

First Season - February - April (summer) with daylight and a mean outside temperature of -17.3°C;

Second Season - May - September (winter) with polar night and mean outside temperatures of -29.3°C;

Third Season - September - January (summer) with daylight and a mean outside temperature of -9.3°C.

Such a classification of the year into seasons, when based on the environmental changes appears over strict; the end of the winter period is dependent upon the termination of the very low outside temperatures rather than that of the polar night; some disappointment is inevitably experienced when the darkness ends but the outside activity remains at a minimum level due to the continuation of these reduced ambient temperatures.

From the description of the changes in the environment at Halley Bay, it is clear that the seasons will have considerable influence upon variables such as that of dietary intake, or energy expenditure. It is relevant at this point to mention the results of

Plate iii. The men could work in low air temperatures with light clothing without discomfort, due to direct and reflected solar radiation.

the detailed work by Norman (1960) on the exposure climates of four subjects at Halley Bay. He reports that the men spent 49% of their time in an exposure climate ranging from 15 to 20°C, and 82% of the time between 0 and 25°C. In effect, this means that at least 82% of the elapsed time was spent within the confines of the base huts, the ambient temperature of which lay between 10 and 20°C. However, these figures represent the average exposure for one year; undoubtedly the exposure is greater in the summer than in the winter months.

Though it has been shown in the Table 2 that the daily mean maximum outside temperatures did not rise above 0°C, the solar radiation was often so marked that the men could work in air temperatures as low as -10°C with bare hands and in only a moderate thickness of clothing without discomfort (see Plate iii). This is not an uncommon situation in the summer months; during severe physical activity, some difficulty can be experienced by expedition members in ridding themselves of excess body heat.

2. The men

The members of the expedition were chosen by reason of their profession, and their physical fitness, or because of their interest in the exploration of unknown territories. The average age of the party was 24.5 years, and the average body weight at the beginning of the year was 74.2 kg. The average height of the men was 179.6 cm. The expedition was composed of six manual workers, eight technicians, nine scientists, and two cooks. This classification is perhaps somewhat unrealistic; there was a considerable overlap of the various duties, many of the men helping in the building and maintenance work which is intrinsic in a community of this nature. Throughout the year, the men lived a routine existence which seldom varied. It is

Plate iv. The newly promoted sledging programme, in which both the energy expenditure and the cold exposure were greatly increased in comparison to base levels.

pertinent to this study to mention that this routine was relieved by two major occupations. Firstly, it was the duty of the men to erect new living accommodation; the project was completed in 6 months but during the first 3 months the work was arduous and exposed. Secondly, the newly promoted sledging programme (see Plate iv) in which the expenditure of energy was greatly increased, as indeed was the cold exposure.

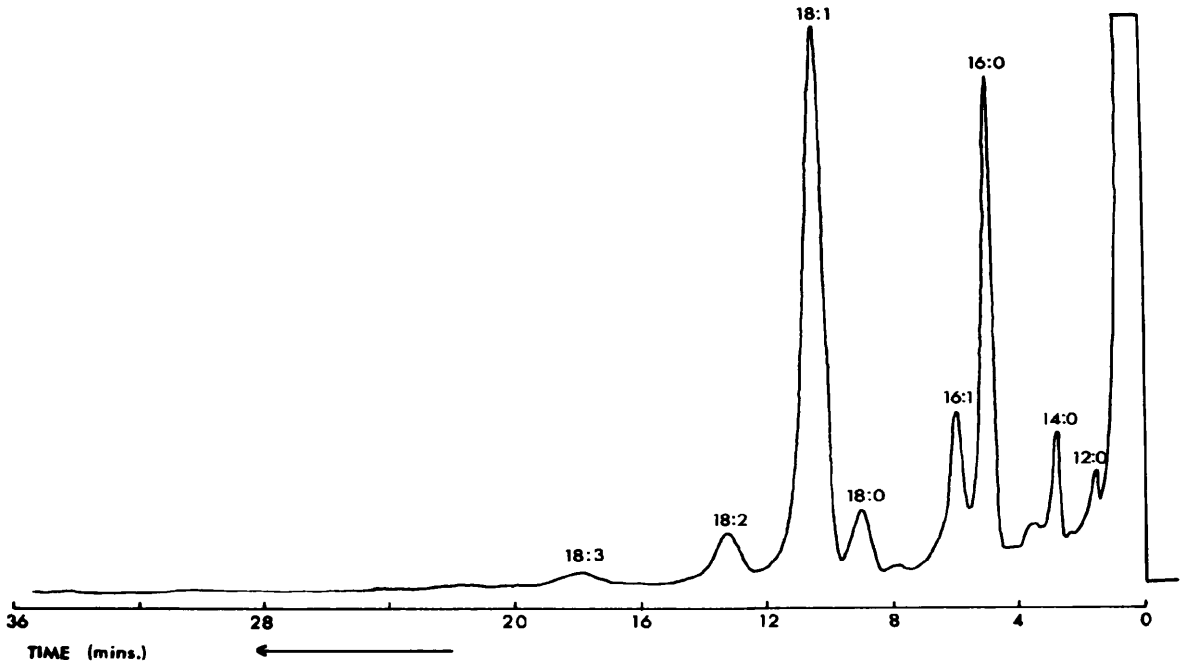
Skin temperatures were assessed on a number of occasions by the author, using a temperature sensitive wire vest (Wolff, 1958) by means of which an integrated temperature of the whole of the trunk could be estimated. On one subject the following skin temperatures were recorded for the ambient temperatures specified: in an environmental temperature of 21.5°C the skin temperature was 32.5°C; in -15°C it was 26.1°C; in -24.6°C it was 26.2°C; and in an outside temperature of -31°C the skin temperature was 22.8°C. The above figures are in each case the mean values of from 10 to 14 original estimations. Prolonged exposure in outside temperatures of -31°C was unusual except during sledging expeditions.

3. The diet

The food at the base was varied and plentiful. Each year stores were ordered on a liberal scale which was always more than adequate. Fruit and vegetables were mainly tinned, though potatoes were also supplied as dehydrated strips and powder. The supplies of tinned meat and fish were supplemented with fresh meat; this allowed a meal of fresh meat on one day/week throughout the year. On the dining room tables vitamin tablets were placed with which many of the men regularly supplemented their diet, although others who did not follow suit showed no evidence at any time of deficiency.

The kitchen was well endowed with all the equipment necessary for the provision of meals for twenty-five men. To facilitate the calculation of the calorific values, frequently recipes were taken from those recommended at the beginning of the M.R.C. report on food values by McCance and Widdowson (1946), in which the calorific equivalents were already known.

Figure 4. A reproduction of a chromatograph of the fatty acids of the adipose fat of subject JN. The fatty acids are marked according to their short hand denomination. Stationary phase: 80 - 100 mesh chromosorb coated with 20% polydiethyline glycol succinate. Temperature = 180°C



SECTION III. VARIATIONS IN THE COMPOSITION OF ADIPOSE FAT

1. Experimental Methods:
   a) In the Field
   b) In the Laboratory
2. Results
3. Discussion
4. Conclusions

Specimens of subcutaneous fat were taken at monthly intervals from twentyfour subjects. The method used was that described by Hirsch, Farquhar, Ahrens, Peterson and Stoffel (1960), by which fat samples are taken from the buttock by a stab biopsy technique. The samples were taken on the same days as the venepunctures, the latter being for the estimation of the serum lipid levels.

1. Experimental Methods

a) In the Field

The skin was initially procainised to produce a bleb. A transfusion needle was then inserted through the procaine wheal to a depth of 2 or 3 cm into the subcutaneous fat layer of the buttock. One millilitre of isotonic saline was then injected from a 50 ml glass venepuncture syringe. Maximum traction was applied to the plunger of the syringe and while suction was maintained the syringe and needle were rotated and pushed back and forth in the adipose tissue. A portion of the injected saline was recovered, and the subcutaneous fat could be seen as minute shiny droplets on the surface of the saline.

The entire aspirate was then quantitatively transferred from the syringe barrel into a 60 ml glass-stoppered bottle by washing the interior with 20 ml of a 1:1 (vol:vol) solution of isopropyl alcohol (Analar, B.D.H.) and petroeum ether (Analar, B.D.H., B.P.30 to 60^{o}C). In order to reduce the risk of oxidative changes 1 µl DL- alpha-tocopherol (Roche) was added to the extracting solution. The samples were stored under deep freeze conditions both on the base and during the return voyage home. Although some evaporation occurred during the year no solutions were completely dried. All bottles were kept sealed with zinc oxide adhesive tape.

b) In the Laboratory

Samples were transmethylated by the micromethod of Stoffel Chu and Ahrens (1959). Each sample was filtered and transferred into a 10 ml test tube; the solutions were then placed in an oil bath at a constant temperature of 60°C, and blown to dryness with pure nitrogen. The dried lipid was then taken up in 3 ml diethyl-ether (Merck) and placed in methylation tubes (16 mm x 150 mm and fitted with a B14 socket). Finally 5 ml 5% H_2SO_4 in pure dry methanol (Analar or Merck) was added with a small piece of porous pot. The mixture was then allowed to reflux in an 80 to 90°C oil bath for 2 hours. Coarse mesh anhydrous calcium sulphate was placed in small drying tubes which fitted tightly into the tops of the methylation tubes. The upper portions of the methylation tubes were cooled by a water condenser system.

After the transesterification, the tubes were allowed to cool, and the contents were neutralised with aliquots of saturated sodium bicarbonate solution. Each specimen was then extracted twice with a few ml redistilled hexane, and was stored in stoppered 10 ml centrifuge tubes in deep freeze until it was analysed by gas-chromatography.

The methyl esters were analysed in a Pye Argon gas chromatograph, utilising an argon ionisation detector with strontium 90 foil (Lovelock, 1958). Two Honeywell recorders (Brown Chart) were used; one as an integrator, and the other for the production of a differential record. Voltages of 1250 or 1500 volts were usually employed, with a sensitivity of x 10 for the differential and x 3 for the integrating record.

The chromatograph was run at temperature of 180 to 181 °C with an Argon pressure of 10 to 15 lb/square inch. The flow rate

was approximately 80 ml/min. A 120 cm glass column (4 mm internal diameter) was filled with 80 to 100 mesh chromosorb on which 20 % polydiethylene-glycol succinate (LAC-2R-728) was coated. A micro syringe (1 µl, Hamilton Co) accurate to 0.01 µl was used for the injection of the methyl esters into the column. Before introduction to the gas chromatograph column, samples were dried over pure nitrogen gas and were then diluted with 40-50 ml redistilled heptane. The Argon used (African Oxygen Ltd) was of high purity and low dew point. The charts were run at a rate of 30"/hr.

The percentage composition of the component fatty acids of the adipose biopsies were calculated by triangulation of the differential record, and by direct measurement of the integral records. Results from the differential and integral records showed close agreement when run together to analyse a standard specimen of methyl esters of fatty acids. Successive runs of the same sample on the chromatograph were found to be accurate to $\pm$ 3%.

It is pointed out that many biopsy samples showed a poor yield of subcutaneous fat and did not produce satisfactory peaks on the chromatographic record. A total of 139 samples of adipose fat were analysed.

2. Results

A typical tracing has been demonstrated in Figure 4 showing the chromatogram obtained from a biopsy of subcutaneous fat. Although all the visible esters on the chart were computed, only the major esters were employed in the assessment of seasonal changes and of individual values. The numbering system for fatty acids, indicating chain length and number of double bonds, is used as suggested by Dole, James, Webb, Rizack and Sturman (1959). The following fatty acids were appraised:

Table 3. The mean fatty acid compositions of the individual adipose biopsy samples for one year, with standard deviations. Only the major fatty acids have been included.

Subject with Nos. of samples		Average percentage of the fatty acids, with S.Ds. Fatty acid short term denomination							
		14:0	14:1	16:0	16:1	18:0	18:1	18:2	18:3
ET	Mn	3.1	1.1	21.9	9.3	4.1	51.3	5.8	2.2
8	S.D.	1.1	0.5	1.4	1.5	1.3	4.1	1.4	0.3
BP	Mn	3.6	0.9	23.6	8.9	5.0	48.2	6.8	2.3
7	S.D.	0.4	0.4	2.3	1.5	0.8	3.7	1.1	0.7
MJ	Mn	3.7	1.6	22.1	11.2	3.7	48.7	6.2	2.2
7	S.D.	0.8	0.1	1.8	1.4	0.9	4.9	0.4	0.4
GT	Mn	2.1	1.1	24.4	5.2	5.4	56.6	4.7	TR
6	S.D.	0.9	0.7	2.7	0.8	1.3	5.3	1.3	-
AP	Mn	3.4	1.3	22.8	9.6	6.0	50.3	5.8	1.5
9	S.D.	1.0	0.4	2.8	1.4	1.3	3.0	0.6	0.4
JS	Mn	3.8	1.1	22.7	9.0	4.6	49.0	6.2	2.1
7	S.D.	1.6	0.4	1.6	1.3	0.6	4.6	1.2	0.4
CD	Mn	2.9	1.2	23.7	8.2	4.1	50.3	6.3	1.4
5	S.D.	0.6	0.5	1.3	0.3	0.9	1.6	0.7	0.3
MS	Mn	4.4	1.3	23.4	10.1	4.3	46.5	6.0	2.6
7	S.D.	0.5	0.5	0.9	1.4	0.7	0.7	0.7	0.3
GM	Mn	3.5	1.6	23.1	9.0	3.2	50.6	6.0	1.8
10	S.D.	0.8	0.8	1.5	1.8	1.0	2.7	0.7	0.6
MTH	Mn	3.8	1.1	23.9	9.3	4.4.	47.2	6.2	2.1
7	S.D.	0.7	0.5	2.2	1.8	1.1	2.9	1.2	0.4
ED	Mn	3.4	1.9	20.9	6.8	4.4	51.5	8.0	1.7
5	S.D.	0.8	0.6	4.1	2.7	1.3	3.2	2.5	1.0
MBR	Mn	4.6	1.4	24.6	8.8	4.8	45.3	7.6	1.7
7	S.D.	0.9	0.4	1.2	0.6	0.7	1.9	1.9	0.3
DJ	Mn	3.8	1.7	23.9	10.0	4.5	46.3	5.8	2.7
7	S.D.	0.8	0.6	1.4	0.7	0.3	2.3	0.5	1.0
PN	Mn	4.4	2.2	22.6	8.6	3.4	48.9	6.2	1.5
6	S.D.	1.3	1.1	0.9	0.4	0.6	4.3	1.4	0.2
SM	Mn	2.3	2.5	20.1	7.6	5.1	48.4	7.1	3.5
10	S.D.	1.1	1.3	2.9	1.7	1.1	5.1	1.4	1.0
GB	Mn	2.9	1.5	21.3	11.3	3.9	46.9	8.9	2.1
6	S.D.	0.9	0.9	1.4	0.5	1.0	3.5	0.4	0.6
RL	Mn	2.7	1.3	20.5	7.5	4.8	52.1	6.9	3.1
11	S.D.	0.9	1.1	2.3	0.9	0.8	4.9	0.7	1.5
DA	Mn	3.9	1.5	23.1	9.2	4.0	48.0	5.5	2.5
8	S.D.	0.8	0.3	1.7	1.2	0.8	3.8	0.8	0.4
DE	Mn	2.6	1.5	22.8	7.9	4.8	49.1	7.2	2.3
6	S.D.	0.9	1.1	2.0	1.7	1.2	3.8	1.6	0.9
Grand Mean		3.39	1.50	22.59	8.83	4.47	49.26	5.89	2.25
Grand S.D.		1.16	0.83	2.44	1.93	1.19	4.52	2.25	0.93

myristic (shorthand designation 14:0), a fourteen carbon fatty acid with one double bond (14:1), palmitic (16:0), palmitoleic (16:1), stearic (18:0), oleic (18:1), linoleic (18:2), and linolenic (18:3). In Figure 4 the fatty acids are labelled according to the nomenclature.

It is stressed that no qualitative check was made on the chemical structure of the component fatty acids. By comparison of the retention times of the fatty acids with those obtained from a standard mixture of fatty acids, surmises were made concerning the structure. In the case of linolenic acid a definite conclusion could not be reached. It will be referred to as linolenic acid in this text, but it should be remembered that it is possible that it may represent a fatty acid of different structure. Linolenic acid seemed the most likely conclusion according to the retention time and the comparison with a standard mixture of fatty acids.

The grand mean for the 139 analyses of fatty acid composition of subcutaneous fat is shown in Table 3 with the means of the individual subjects. The standard deviation of the grand mean was in the case of palmitic and oleic acids respectively 2.44 and 4.52 mg %. Hence in the case of palmitic acid, the range (that is 95% of all results) lay between 17.8 and 27.4%, and oleic acid was distributed between 40.4 and 58.1%. It is seen that standard deviations of the six remaining fatty acids measured were high. Perusal of Table 3 shows that there were inter-individual differences in subject mean fatty acid concentrations. It also indicates that there was a certain degree of intraindividual variation

The samples were examined for seasonal trends. Samples taken within each season were grouped together, and the individual means were calculated (see Tables 39, 40 and 41 in the Appendix). The seasonal means for the whole group are shown in Tables 4 and 5. That all the percentages do not add up to 100 exactly was due to the elimination of

Table 4. Shows the changes in fatty acid composition of subcutaneous fat for the three seasons of the year.

Season	Fatty acid short term denomination 14:0	14:1	16:0	16:1	18:0	18:1	18:2	18:3
Feb./ April	3.5 ±1.1	1.5 ±0.6	23.2 ±2.2	8.9 ±2.0	4.8 ±0.8	47.6 ±4.1	6.7 ±2.0	2.1 ±0.7
May/ Sept.	3.4 ± 0.8	1.4 ±0.6	22.6 ±1.7	9.3 ±1.5	4.2 ±0.9	49.2 ±2.9	6.7 ±0.9	2.1 ±0.6
Oct./ Jan.	3.2 ±0.9	1.4 ±0.6	22.1 ±2.6	8.1 ±2.0	4.5 ±2.7	50.4 ±4.2	6.5 ±1.6	2.4 ±0.8

trace acids such as lauric acid and C 17:0 fatty acid.

It is seen that oleic acid increased by 2.8% and that palmitic acid decreased by 1.1% between the first and the third seasons. Both myristic and stearic acids also tended to fall, but there was a rise in stearic from the second to the third season of 0.3%. The remaining unsaturated fatty acids (14:1, 18:2 and 18:3) demonstrated clearly non-significant changes. Palmitoleic acid, however, showed a fall of 1.2% from the second to the third season, which was preceded by a rise of 0.4% between the first and the second seasons. The seasonal change which occurred in the total saturated fatty acids was small, and there was no clearly defined trend. The total unsaturated fatty acids showed a slight rise in the second and third seasons.

The individual seasonal means of oleic acid levels were examined by variance analysis, and both the 'between men' effect and the 'between seasons' effect proved to be significant at the 0.5% and the 5% levels respectively. Similar treatment of the total unsaturated fatty acids showed the 'between men' effect to be significant at the 0.1% level, and the 'between seasons' effect to be significant at the 5% level. For the total saturated fatty acids, significance could only be established at the 5% level for the 'between men' effect and the 'between seasons' effect was non-significant. The relevant data to these analyses is tabulated in Tables 42, 43 and 44 in the Appendix.

Using the appropriate comparison technique, for oleic acid the difference between the first and the second seasons was significant at the 2% level, and the difference between the first and the third seasons was significant at the 0.1% level. The difference between the second and the third season was non-significant. In the case of the total unsaturated fatty acids the difference between the first and

Table 5 . Seasonal changes in adipose biopsies in total saturated fatty acids, total monounsaturated and total unsaturated fatty acids.

Season	Total Saturated	Total Monounsaturated	Total Unsaturated
Feb/April	30.0	58.0	66.8
May/Sept.	30.2	59.8	68.6
Oct/Jan.	29.8	59.9	68.4

second seasons and between the first and third seasons were both significant at the 0.1% level. It is apparent from these results, therefore, that there was a significant rise between the first (summer) and the second (winter) seasons in both oleic acid and the total unsaturated fatty acids of adipose fat.

In the case of the individual mean values (Table 3) there was no correlation between the elevation and depression as a percentage of any pair of fatty acids. For example, a high level of oleic acid was not conspicuously associated with a depression in palmitic acid, or any other fatty acid estimated. The average individual total of unsaturated fatty acids in those subjects who had the greatest outside exposure (GT, RL, MTH and DA) was not elevated over the mean level for the remaining group of subjects.

3. Discussion

In Table 6 the grand mean of all observations made during the Halley Bay survey has been grouped for purposes of comparison with data recorded by other workers. The adipose biopsies from this survey agree most closely with the findings of Hegsted, Jack and Stare (1962) on biopsy material taken from subjects living in Boston, Mass. They differ from the findings of Hirsch et al (1960) who studied a group of twelve normal males and females in New York. The mean level of linoleic acid is lower in the Halley subjects, while palmitic and oleic acids are higher. It is unlikely that any of the differences shown in the fatty acids would be significant. It is seen that there is a large variability in any one population group. Hegsted and his co-workers tested the difference between the Japanese, Columbian and Jamaican groups with the three Nigerian groups and found no significance for the major fatty acids.

Table 6. The fatty acid composition of the Halley Bay subjects compared with surveys made in other geographic regions.

Regions	Fatty acid short term denomination							
	14:0	14:1	16:0	16:1	18:0	18:1	18:2	18:3
American Males & Females from New York (Hirsch et al, 1960)	3.3 ±0.1	0.6 ±0.1	19.5 ±2.1	6.9 ±0.1	4.2 ±1.1	46.3 ±4.4	11.4 ±1.4	0.4 ±0.1
Halley Bay Subjects	3.4 ±1.1	1.5 ±0.8	22.6 ±2.4	8.8 ±1.9	4.5 ±1.2	49.3 ±4.5	5.9 ±2.3	2.2 ±0.9
Bostonians (Hegsted, Jack & Stare, 1962)	3.6 ±1.0	TR -	24.6 ±3.0	6.1 ±2.0	6.7 ±1.8	50.3 ±3.4	7.9 ±3.5	TR -
Japanese (Hegsted et al, 1962)	3.4 ±1.6	0.8 ±0.3	24.9 ±4.9	10.5 ±2.8	4.8 ±2.2	40.6 ±4.2	9.4 ±3.5	
Columbians (Hegsted et al, 1962)	4.3 ±0.9	0.5 ±0.4	25.1 ±1.1	8.5 ±2.1	6.9 ±1.5	45.7 ±4.1	5.5 ±1.1	
Jamaicans (Hegsted et al, 1962)	8.4 ±2.0	1.4 ±0.6	26.6 ±2.9	10.1 ±2.6	5.8 ±1.8	37.7 ±3.2	5.8 ±2.0	
New Yorkers (Scott et al, 1962)	4.1 -	- -	25.0 -	6.3 -	4.3 -	50.2 -	9.5 -	
Nigerians								
Group A	- -	- -	26.2 ±3.7	6.7 ±2.5	9.6 ±2.6	42.3 ±3.4	8.7 ±3.7	
Group B	- -	- -	28.5 ±2.9	5.9 ±1.8	7.1 ±1.3	46.2 ±3.7	7.9 ±1.8	
Group C	- -	- -	28.7 ±2.5	5.4 ±2.1	8.0 ±1.7	46.1 ±2.5	8.0 ±2.2	
(Hegsted et al, 1962)								

The standard deviations for all fatty acids ascertained from the subjects stationed in the Antarctic were numerically similar to the standard deviations from the other survey results presented in Table 6. The interesting feature of the fatty acid components of the Halley Bay group was the maintained existence of linolenic acid in more than trace quantities. This has been reported by Hirsch et al (1960) when it amounted to less than 1% of the total acids.

Kingsbury, Paul, Crossley and Morgan (1961) investigated the composition of the adipose fat in a group of English subjects using gas-chromatography. They found that depot fat contained 42-51% of oleic acid, 21-30% of palmitic acid, 5-8.5% of palmitoleic and stearic acids, 5.8% of linolenic acid, and under 3% of acids with more than two double bonds. The total saturated fatty acids and the total unsaturated fatty acids amounted to 37.5 and 59.3% respectively. The Halley Bay figure for the total of unsaturated fatty acids was 67.7% which was considerably higher than the result reported by Kingsbury and his group, which is of some interest. The greater the degree of unsaturation the lower the melting point, and if the group of Kingsbury et al can be accepted as typical of the United Kingdom, this difference suggests a lower adipose fat melting point in Antarctic personnel.

Kingsbury et al noted that no significant differences were found between the depot fat composition of normal subjects and of patients with severe atherosclerosis, excepting possibly an increased concentration of triunsaturated acids, particularly in relation to dienes and tetraenes. Thus in normal subjects the mean level of linolenic acid was 0.45% and in atheromatous subjects the mean level was 0.96%. Yet this latter result still does not compare with the mean level of linolenic acid in the Halley Bay group, which amounted to a mean of 2.2%. The possibility

exists that the elevated level of linolenic acid was a dietary phenomenon, or may have been an effect of exposure to cold stress.

It is clear, from Table 3, that the results of the analysis of the fat biopsies of each subject showed an intra-individual variation which in most cases was similar to the standard deviations of the grand mean for all samples. This was the cardinal feature of the individual subcutaneous fat compositions; in men who were consuming the same basic diet, each individual demonstrated a remarkable fluctuation of levels for all fatty acids measured.

That the variation which has been shown might have resulted from a complication of the biopsy technique used in this survey is possible. There was no method of estimating the true depth beneath the cutis at which the fat was taken. It has been reported that in subcutaneous tissue, the iodine number of the fatty acids decreases and the fat becomes more saturated with the depth from which the biopsy is taken (Henriques and Hanson, 1901; Wertheimer and Shapiro, 1948). However, Cuthbertson and Tompsett (1933) reported that these differences are negligible in man. Hirsch et al (1960) compared the composition of fatty tissue removed from various deep sites with a needle biopsy of the subcutaneous fat taken from the buttock. Only in the case of fat taken from the pericardium and psoas muscles was there a lesser degree of unsaturation, and in the cases of biopsies taken from the omental and perinephric fat, the percentage of unsaturated fatty acids was slightly increased. Hence it seems that the depth below the cutis from which the specimen was taken would not influence the composition from the results of the latter two groups of workers.

The degree of variation found in the major subcutaneous fatty acids in these subjects is a decisive indication that the adipose organ is in a constant state of flux. From the results of the Halley

Bay survey no estimation of the rate of change of the component fatty acids can be computed; they merely act as an indication. Further investigations, in which samples of subcutaneous fat taken at shorter intervals of time, would provide an answer concerning the rates of change of the fatty acid compositions.

Until recently the usual idea of the function of the adipose organ was that it was a static energy store, taking up surplus energy under conditions of excess eating, storing it in the form of fat, and giving it up when the food intake was too little.

Keckwick (1960) has summarised evidence which suggests that the adipose organ plays a more than inactive role in metabolic processes in the human. His statements were based on in vitro and in vivo animal studies. The rate of turnover of deuterium and radioactive carbon labelled fatty acids in normal intact mice and rats indicated that half the adipose organ was metabolised and reconstituted in 5 to 9 days. Though no measurements of turnover have been made using labelled fat in the human, Keckwick using indirect evidence based on the occurrence of normal B.M.Rs in the obese, argued that if fat were metabolically inactive the oxygen uptake of an obese subject should be comparable with that of a lean subject. The fact that this was not so prompted the conclusion that the adipose organ was metabolically active, and required the extra oxygen. Keckwick summarised by pointing out that there is indirect evidence in man, and direct evidence in animals, that the adipose organ is metabolically highly active with a turnover rate far more rapid than can be accounted for in terms of fat and carbohydrate intake.

Yet Hirsch et al(1960) in a study of the effects of a high intake of corn oil in the diet of eight subjects reported that changes in the adipose organ were almost imperceptible up to 20 weeks. However,

the adipose tissue in these subjects resembled a mixture of corn oil and normal adipose fat in a proportion of 7 to 3 at 160 weeks. Hirsch and his co-workers calculate a half life for linoleic acid as being between 350 and 750 days. They reconcile these findings with the postulate that two separate metabolic compartments exist in the adipose fat; a large one which might serve as an inert storage for fat calories, and a much smaller compartment with a rapid turnover in close relation to dietary, serum and liver lipids.

The variability of the individual lipid levels in the Halley Bay group does not agree with the findings of Hirsch et al but accords with the suggestions of Keckwick.

The seasonal trends of the total unsaturated fatty acids were significant. It is seen from Table 5 that the total unsaturated acids rose by 1.8% from the first to the second seasons, and fell by 0.2% from the second to third seasons. It is of interest to calculate the change in melting point accountable to this alteration in the degree of unsaturation. Ellis and Hankins (1925) in their soft pork studies showed that a decrease in the percentage of unsaturated fatty acids from 66.9 to 61.4 was associated with a rise in melting point from 25.5 to 37.5°C. It would follow that a change of 5.5% in the percentage of unsaturated fatty acids would account for a rise or fall in melting point of 12°C. Hence a change of 1% in saturation of the component fatty acids would be equivalent to 2.2°C, and a change of 1.8% would be equivalent to almost 4°C rise or fall in melting point, and from this it could be implied that the Halley Bay subjects showed a fall of 4°C in the melting point of their subcutaneous fat between the first and second seasons. Fair criticism can be levelled at a deduction made from animal experiments. However, it must be emphasised that the subcutaneous fat of the pig shows close similarity in fatty acid pattern

to the composition of the adipose layer in man.

Interest and popular scientific opinion have varied over the relative importance of environmental factors in their influence on the composition of the adipose fat. The suggestion that it is the degree of unsaturation of the fatty acid components of the serum lipids which determines their serum levels and that this composition might somehow be mirrored in the subcutaneous fat, has caused renewed investigation, particularly in relation to patients with atheromatosis or overt coronary thrombosis (Penman, 1960; Kingsbury, Paul, Crossley and Morgan, 1961; Hegsted, Jack and Stare, 1962; Scott, Daoud, Gittelson, Opalka, Florentin and Goodall, 1962; Kingsbury, Morgan, Aylott, Burton, Emerson and Robinson, 1962). Most of these investigators have returned negative conclusions; the chemical composition of the adipose organ shows little positive relation to the degree of arterial atherosclerotic change. From the changes shown in the Halley Bay survey, it might be hypothesised that the fatty acid composition of the adipose fat depends to a large extent upon the environmental temperature.

There is a certain amount of data which has been collected over the years substantiating this hypothesis. Reference has already been made to some of the earlier investigations in the introduction of this report. Henriques and Hansen (1901) in studies of the pig demonstrated an inverse relationship between environmental temperature and the extent of depot fat saturation. This relationship between depot fat and tissue temperature might have been due to the relative lack of tissue insulation in the pig. However, recently Fisher, Hollands and Weiss (1962) have investigated the influence of variations in ambient temperature on the composition of the subcutaneous fat of the chicken and shown positive results. The authors stated that there were

no changes in deep body temperatures or temperature of the tissue at the site of the fat biopsy which correlated with the changes in fat composition. They suggested that metabolic adjustments associated with cold adaptation were responsible for the change in body fat composition. Hence, it is clear that the changes which occurred in the pig were not merely due to the fact that the pig has a relative lack of body insulation.

Young and Cook (1955) examined the effect of high and low temperatures on the carcass fat of rats and found that the melting point was proportional to the temperature of adaptation. Mefferd, Nyman and Webster (1958) fractionated fat from rats exposed to cold, control and hot environments into solid and liquid fatty acids by the lead-soap precipitation method and they found that the proportion of liquid fatty acids decreased linearly with temperature.

Kodama and Pace (1963) have measured the composition of tissue fat in hamsters during acclimatization to cold. Their findings were almost exactly similar to those at Halley Bay, the animals showing a rise of oleic acid and a fall of palmitic as they became acclimatized. As well as occurring in the subcutaneous fat, changes very similar in magnitude also were apparent in peri-renal fat. The authors suggest therefore that the increase of unsaturation was a generalised phenomenon. In an extension of the experiment, semi-starvation and administration of tri-iodothyronine plus cortisone both had effects similar to those of acclimatization. It was concluded that the increase was due to the preferential use of saturated acids, with residual accumulation of unsaturated fatty acids. The authors finally suggest that the changes may well be one of the indices of acclimatization.

McDonald (1961) investigated the subcutaneous fat of infants in the Cape Province, and compared them with samples taken in East Africa. He showed that there was a rise in iodine number as a function

of age in years in the South African infants but that no rise occurred in the East African children. McDonald concluded that the results were consistent with the hypothesis that the iodine number of depot fat is determined, to some extent, by the ambient temperature. McLaren and Read (1962) came to a similar conclusion in attempting to explain the similarity of compositions of adipose biopsies from three racial groups in East Africa, who were consuming dietary fat which was very different both in composition and in the proportion of the total calorie intake which it supplied.

It may be that racial differences as determined by Hegsted, Jack and Stare (1962) and others are dependent on ambient temperatures to a greater extent and not so much on the composition of the dietary fat. The Halley Bay findings would substantiate such a view.

It seems unlikely that the increase of the degree of unsaturation of the adipose fat was related to the gain in body weight which occurred in the first 2 months of the year (see Section IV). The soft pork studies of Ellis and Hankins (1925) showed that weight gain was associated with hardening of the adipose fat. It also appears unfeasible that a small fall in the melting point of the subcutaneous fat would affect the body insulation. It is probable that the important governing factor is that of joint mobility; a hard adipose fat at low temperatures would clearly be a disadvantage.

It was concluded in the introduction that due to efficient insulation, the human in polar regions is not subjected to severe cold stress, and shows few physiological changes. However, it is apparent that the cold stress is great enough to influence the fatty acid composition of the adipose fat. Whether it is a generalised phenomenon, or whether the unsaturation changes as a function of the degree of

localised cold stress (i.e. would it be greater in the adipose fat of the face or extremities?) is clearly of great interest and worthy of further research.

4. Conclusions

From the survey of adipose fat in the men at Halley Bay, the following conclusions may be drawn:-

i. The grand mean values of the fatty acid expressed as a percentage of the total were:
myristic 3.4%; palmitic 22.6%; palmitoleic 8.8%; stearic 4.5%; oleic 49.3%; linoleic 5.9%; and linolenic 2.2%

ii. That the mean individual values of fatty acids composing the adipose fat were scattered over a wide range. This is of particular interest as all members of the group were consuming the same diet.

iii. That the estimations of the composition of subcutaneous fat for separate individuals suggest that the adipose organ is labile and fluctuating.

iv. That there was a high content of linolenic acid in the adipose fat of the subjects, which has not been reported in amounts of more than 1% by previous workers.

v. That the seasonal change suggested from the means of the oleic acid and the total of unsaturated fatty acids was statistically significant. The increase of unsaturation shown in the samples would account therefore for a fall in melting point of the adipose organ. The possibility exists that the changes in the degree of unsaturation found during a year's stay in the Antarctic may represent an index of acclimatization in man.

SECTION IV. VARIATIONS IN BODY WEIGHT, SKINFOLD THICKNESS AND FOOD INTAKE

1. Experimental Methods

2. Results

   a) Body weight

   b) Skinfold thickness

   c) Dietary intake

3. Discussion

   a) Body weight

   b) Skinfold thickness

   c) Dietary intake

      i. Total Calories

      ii. Protein

      iii. Fat

      iv. Carbohydrate

   d) The energy requirements of four occupational groups

   e) General discussion

4. Conclusions

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1. Experimental Methods

The subjects were weighed at monthly intervals without clothing before breakfast, having passed urine. A 150 kg Steelyard Bench Platform Scale (Avery) was used, which was accurate to the nearest 50 gm.

At the same time as the estimation of the body weights, the skinfold thickness was measured at five sites with the Harpenden spring loaded calipers as described by Edwards, Hammond, Healy, Tanner and Whitehouse (1955) at a pressure of 1020 gm. The five sites suggested by Lewis, Masterton and Ferres (1958) as being the most accurate were used, viz.

I. A point midway between the nipple and the axillary fold.
II. A point 6 inches above the head of the radius, on the lateral side of the upper arm.
III. A point over the inferior angle of the scapula.
IV. The midpoint of a line between the umbilicus and the anterior superior iliac spine.
V. A point 9 inches above the head of the fibula, on the lateral side of the thigh.

Each site was measured five times with the calipers and the average result for each site was employed in the final analysis. In the following pages the sites will be referred to according to the above numerical denomination. A fold of skin was picked up between the finger and thumb and the calipers were directly applied; the skin thickness value was recorded in mm when the needle had reached a constant value. In some subjects there was some difficulty associated with the measurement made on the lateral side of the thigh. The skin tends to be taut in this region, but often by complete relaxation of

the thigh (the subject was requested to be supine) a reading could be recorded. On subject RL however it was impossible to gain a result and so the site on this subject was omitted for the year. Also the pectoral site on subject GB was ignored due to the difficulties associated in obtaining a skinfold as a result of fascial attachments.

It should be stressed that there are certain disadvantages connected with making these measurements. It is often difficult to find the same site each time a measurement is made. There does not appear to be a method whereby the skin can be marked for a period of up to a month; needless to say many agents were investigated, but the only method suitable was that of tatooing the skin, and the author did not feel that volunteers should undergo this as well as the other somewhat distasteful operations to which they were subjected. A second difficulty is associated with the movement of the needle on the dial; on applying the calipers the needle slowly falls until a constant value is reached for a few seconds, after which the reading continues to fall. It was the practice to take the reading when the needle ceased to move the first time. However, difficulty was encountered when the needle failed to demonstrate the preliminary levelling.

The dietary survey was made by the direct method of weighing the total food intake over 24 hours and the calorific values were allotted from the tables of McCance and Widdowson (1946). During the year, each of the twentyfive subjects on the expedition had a food intake assessed eight times in all. It took approximately 6 weeks to estimate the dietary intake of the whole group, the consumption of four subjects being assessed every week. Towards the end of the year, however, due to the interference of other work, three subjects were assessed on the survey days so that in effect the interval was

shortened to last four or five weeks.

On the survey days the subjects were provided with cards on which they recorded the amount of food eaten apart from the official meals of the day. Much of this was consumed during three tea breaks in the morning, afternoon and evening, and was carefully weighed and measured by the volunteers.

The intakes of the three main meals were recorded on stencilled sheets and were weighed out in the kitchen before the meal began. On separate forms, the composition of the recipes which composed the meal was noted. The prepared dishes were weighed before and after cooking and the loss or gain in weight accounted for in the final calculation of the calorific values. All the food eaten in the dining room was weighed on a dietary balance to the nearest 2 gm. Waste from the plates was removed by scraping and was weighed and noted for the final assessment. Foods such as sugar and milk in tea, and confectionary goods were estimated from previous weighings and the correct quantity allotted from the numerical description.

The data on the private cards were added to the proformata on which the intake of the three main meals were recorded. Having calculated the values of the recipes, the complete dietary intakes for two subjects over 24 hours was assessed as protein, fat and carbohydrate in grams and as total calories. The food values of most of the products on the base were obtained from the tables of McCance and Widdowson (1946) and some of the more unusual tinned foods from figures provided by the Army Catering Corps.

Over the year 200 estimations of dietary intake were made (see Tables 65-72 in the Appendix).

Figure 5. The scattergram of the individual changes in body weight of subjects domiciled at Halley Bay for one year. The continuous line represents the monthly mean of all subjects, and the interrupted lines are the standard deviations.

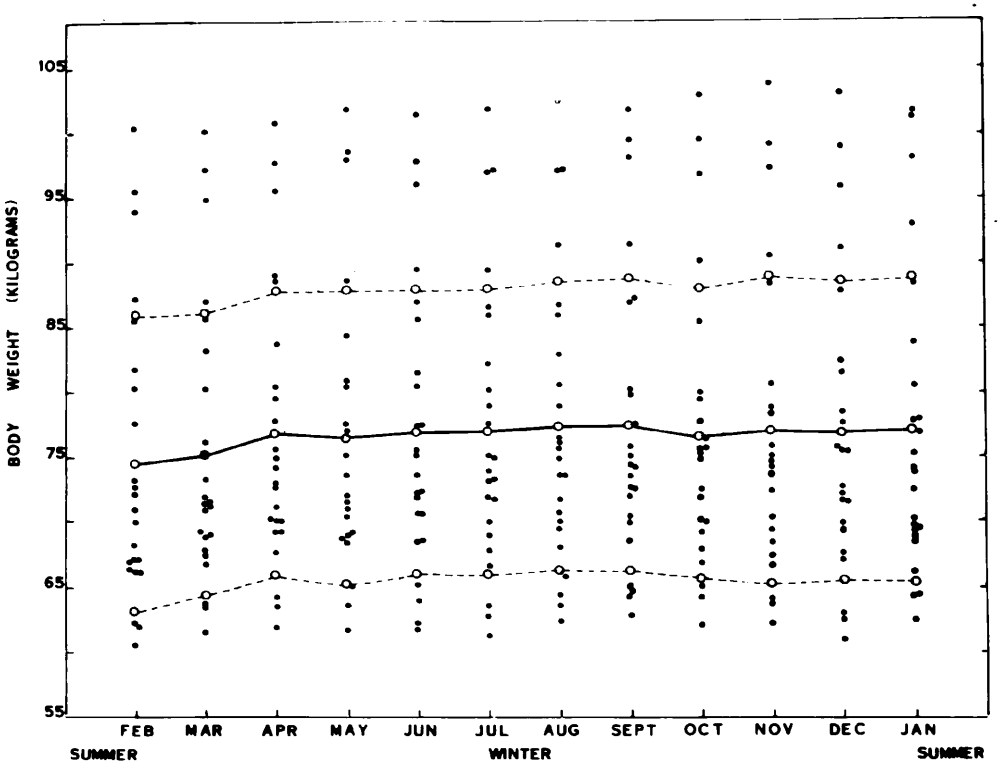


Table 7 . Mean monthly weight changes for 24 men - in Kg

	Feb	Mar	Apr	May	Jne	Jly	Aug	Sep	Oct	Nov	Dec	Jan
Mean body weight	74.3	75.2	76.8	76.6	76.9	77.1	77.4	77.4	76.9	77.1	77.0	77.0
Standard deviation	±11.1	±10.5	±10.8	±11.1	±10.9	±10.9	±10.9	±11.0	±10.9	±11.6	±11.3	±11.4

2. Results

a) Body Weights

The monthly changes in body weights are demonstrated as a scattergram in Figure 5, and the meaned monthly values with standard deviations are shown in Table 7. The subjects gained weight at a maximum rate during the first 2 months of the year (February to April) and then they levelled off and remained at a constant level for the rest of the year. There was a very slight tendency to lose weight at the end of the year. The scattergram (Figure 5) shows that the body weights were distributed over a wide range.

b) Skinfold Thickness

The mean monthly changes in skinfold thickness have been summarised graphically in Figure 6 and have been tabulated in Table 8. To obtain a gaussian distribution of the results, the values of the measurements of the skinfold thickness in mm were converted to a log transform as proposed by Edwards, Hammond, Healy, Tanner and Whitehouse (1955).

$$\text{Transformation} = 100 \log_{10} (\text{reading in } 0.1 \text{ mm} - 18)$$

In Figure 6 the points shown represent the average values for the log transform of the skin thickness for each site, the thickness having been assessed five times for every original reading. Table 8 shows the average results of the log transforms, with standard deviations, and the means are also demonstrated in the original units of their measurement.

Due to the size of the group of subjects taking part, each skinfold thickness site could be examined for a monthly effect by analysis of variance. Subjects GB, DE, CJ, AM, ET and MTH were eliminated from the analysis in order that a complete block could be obtained in which

Figure 6. The monthly changes in the 5 skinfold thickness sites, demonstrated as the log transform. Each point is the average of 24 readings.

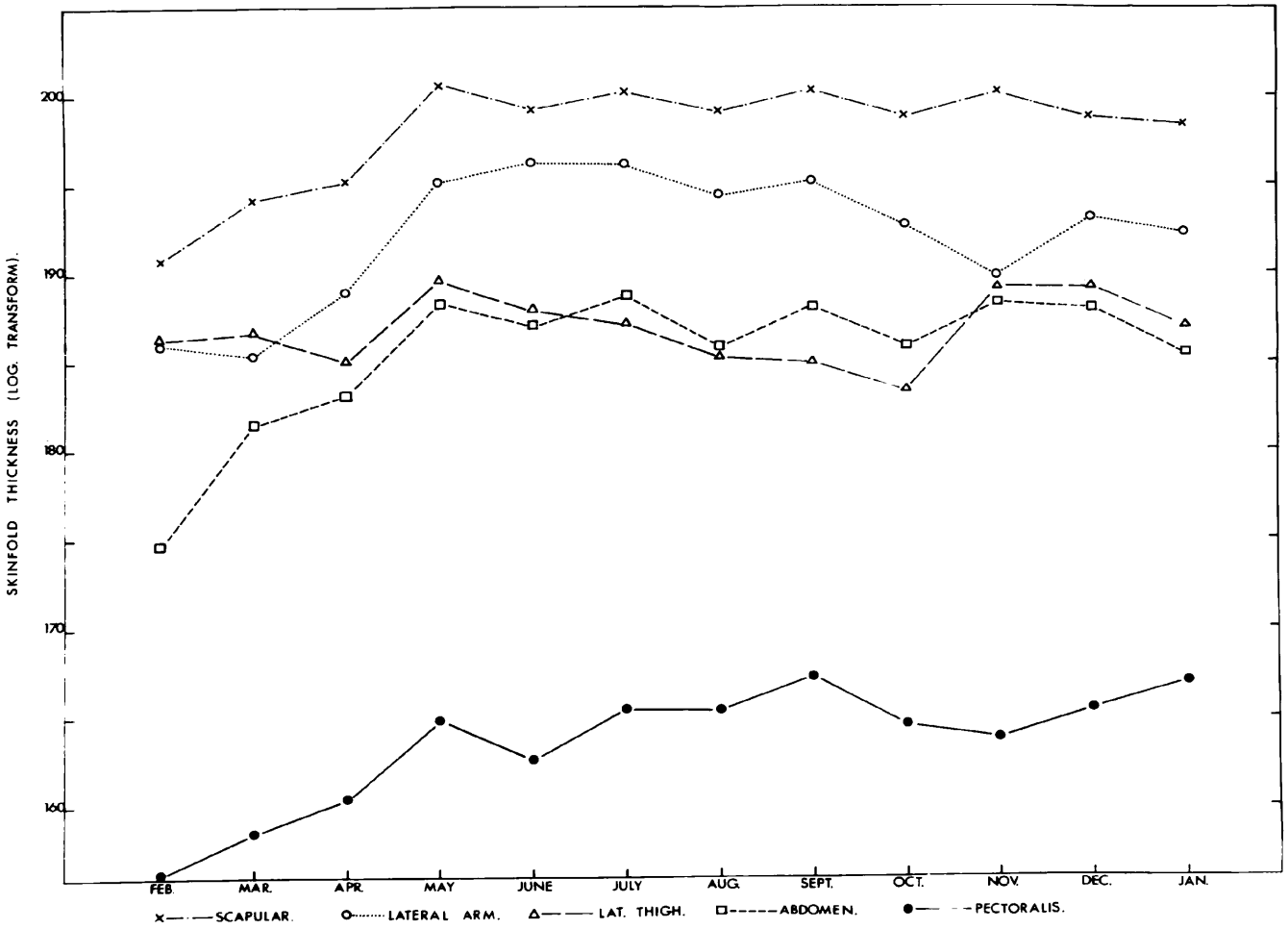


Table 8. The monthly mean skinfold thickness values for five sites, demonstrated as the log transform (with standard deviations) and as the absolute values in mm.

	Log Transform					Absolute value (mm)				
	Site					Site				
	I	II	III	IV	V	I	II	III	IV	V
February 1961	156.0	186.0	190.7	174.7	186.2	5.4	9.0	9.8	7.3	9.0
SD	21.1	9.3	19.5	23.9	18.9					
March	158.5	185.4	194.2	181.5	186.7	5.6	8.8	10.5	8.3	9.1
SD	24.0	20.7	14.6	20.5	24.2					
April	160.4	189.0	195.3	183.2	185.1	5.8	9.6	10.8	8.6	8.8
SD	23.1	16.7	14.8	22.3	21.4					
May	164.9	195.2	200.7	188.4	189.7	6.3	10.7	11.8	9.3	9.6
SD	26.3	17.8	21.7	25.5	31.3					
June	162.6	191.3	199.3	187.1	188.0	6.0	9.9	11.5	9.2	9.3
SD	27.0	20.7	16.7	23.6	19.9					
July	165.5	191.2	200.3	188.8	186.2	6.3	9.9	11.7	9.4	9.0
SD	25.5	19.0	17.2	28.4	25.2					
August	165.4	194.5	199.2	185.9	185.3	6.3	10.6	11.5	8.9	8.8
SD	24.5	22.4	14.9	28.5	24.7					
September	167.3	195.2	200.4	188.2	185.0	6.5	10.7	11.8	9.3	8.8
SD	23.7	17.7	17.8	23.6	23.9					
October	164.6	192.7	198.9	185.9	183.4	6.2	10.2	11.4	8.9	8.5
SD	27.7	31.5	23.2	22.0	27.2					
November	163.8	189.9	200.3	188.4	189.3	6.1	9.6	11.7	9.3	9.5
SD	28.3	27.0	17.9	24.5	24.7					
December	165.5	193.1	198.9	188.0	189.3	6.3	10.3	11.4	9.3	9.5
SD	34.8	33.5	24.2	21.4	23.5					
January 1962	167.0	192.3	198.4	185.5	187.0	6.5	10.1	11.3	9.4	9.2
SD	23.4	18.2	18.2	25.9	20.0					

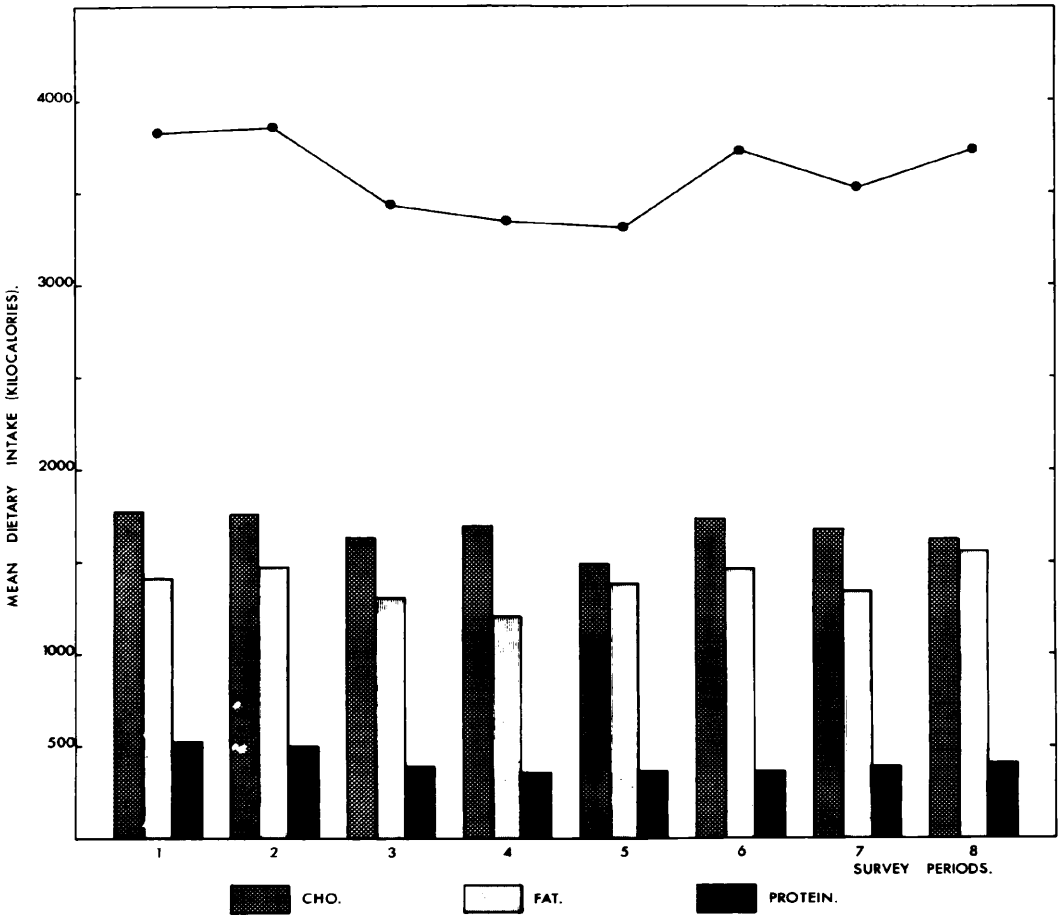
there was a reading for each person for each month. In the case of the analysis of the measurements made on site V, subject RL was also eliminated.

A 'between months' effect and a 'between men' effect were found to be significant at the 0.1% level for skinfold thickness sites I to IV, but significance was not established for site V, the measurement made on the lateral side of the thigh. The non-significant result could be explained partially by the difficulty in finding exactly the same position each time the measurements were made; the thickness of the subcutical fat varies quite considerably in the circumferential plan of the thigh. It is seen from Figure 6 that the subjects increased the skinfold thickness at sites I, II, III and IV from the first month of the experiment up to the third month. This is of great interest as the body weight mean had levelled by April. After the period of increase in skinfold thickness, the sites maintained a comparatively constant level for the remaining months. Comparison tests showed that the difference in the mean skinfold thickness expressed as the log transform, between the months April and May for site I was significant at the 1% level, while for sites II, III and IV, the differences were significant at the 0.1% level.

c) Dietary Intake

The results of the dietary intake survey are shown in Tables 9, 10, 11 and 12 and Figure 7. For the purposes of statistical analysis, the survey was divided into eight periods during the year, and there was a single estimation of caloric intake for each person in each period. The periods were initiated and completed on the following dates:

Figure 7. The changes in the dietary intake of total calories, and fat, carbohydrate and protein calories for each period of the diet survey.



	Grand Mean	Percentage
Protein	426 ± 135	12.1
Fat	1403 ± 454	39.8
CHO	1691 ± 510	48.1
Total Cals	3597 ± 929	

Table 9. Grand means with percentage composition for food intake/man/day, from 200 assessments, in calories.

Period I	13. 2.61 - 26. 3.61
Period II	29. 3.61 - 9. 5. 61
Period III	12. 5.61 - 16. 6.61
Period IV	25. 6.61 - 1. 8.61
Period V	2. 8.61 - 11. 9.61
Period VI	15. 9.61 - 9.10.61
Period VII	13.10.61 - 22.11.61
Period VIII	30.11.61 - 27.12.61

In Figure 7 and Table 10 the mean levels of total calorie intake and protein, fat and carbohydrate intakes have been demonstrated. It is seen that all levels were depressed during period III, IV and V, when compared with levels in periods I and II, and in periods VI, VII and VIII. The periods III, IV and V were regarded as representative of the winter food intake values. Thus, three unequal blocks resulted: periods I and II comprising the first summer season, III, IV and V making up the winter values, and VI, VII and VIII being allotted to the third season which was the second summer season. With the use of an analysis of variance technique involving the use of these blocks, the total calorie intake and the consumption of protein and fat in calories were found to be significant at the 0.1% level for a 'between seasons' effect, and,with the exception of the protein intake (which was non-significant), were also significant for a 'between men' effect at the 0.1% level. The carbohydrate intake was significant at the 1% level for a ' between seasons' effect, and at the 0.1% level for a'between men' effect. The relevant data is shown in Tables 78 - 81 in the Appendix.

The weighted means of the three seasons (Table 12) were examined by a comparison technique and the significance was found to be as follows:

Table 10. Means for total calories and three food groups in calories with Standard Deviations, for each dietary survey period

	I 13:2-26:3	II 29:3- 9:5	III 12:5-16.6	IV 25:6- 1:8	V 2:8-11:9	VI 15:9-9:10	VII 13:10-22:11	VIII 30:11-27:12
Total Cals	3824	3863	3434	3346	3301	3729	3527	3733
	±836	±1104	±813	±844	±934	±1067	±892	±821
Protein Cals	528	501	392	362	366	367	389	413
	±156	±187	±105	±101	±99	± 99	±124	±413
Fat Cals	1417	1475	1307	1207	1381	1464	1343	1560
	±448	±614	±429	±337	±503	±502	±517	±400
CHO Cals	1774	1759	1633	1699	1494	1734	1679	1623
	±406	±500	±364	±506	±479	±489	±449	±370

Table 11. Mean percentage compositions of dietary intakes for each of the eight survey periods

	I	II	III	IV	V	VI	VII	VIII
Protein %	14.2	13.4	11.8	11.1	11.3	10.3	11.4	11.5
Fat %	38.1	39.5	39.2	36.9	42.6	41.1	39.4	43.4
CHO %	47.7	47.1	49.0	52.0	46.1	48.6	49.2	45.1

Total calories

Season I compared with season II was significant at the 0.1% level
Season I compared with season III was non-significant
Season II compared with season III was significant at the 5% level

Protein calories

Season I compared with season II was significant at the 0.1% level
Season I compared with season III was significant at the 0.1% level
Season II compared with season III was non-significant

Fat calories

Season I compared with Season II was significant at the 1.0% level
Season I compared with season III was non-significant
Season II compared with Season III was significant at the 1.0% level

Carbohydrate calories

Season I compared with season II was significant at the 1% level
Season I compared with season III was just non-significant
Season II compared with season III was non-significant

Thus the depression in total calorie intake in the winter months was significant. The fall in food intake from the first summer to the winter occurred in protein, fat and carbohydrate intakes but the rise from the winter to the second summer period was largely due to a rise in fat intake. The excess of calories in season I over that in season III was due to an increased consumption of protein and carbohydrate and not of fat calories.

Table 11 shows the percentage of calories supplied by protein, fat and carbohydrate for each period of the dietary survey. Apart from the tendency for the protein consumption to provide a greater proportion of

Table 12. Weighted means for totalcalories and the three food groups in calories with the percentage compositions for the three seasons. Periods I & II comprise the first season; III, IV & V the second; and VI, VII & VIII the third.

	Summer	Winter	Summer
Protein	521	373	390
	13.9%	11.4%	11.1%
Fat	1458	1298	1455
	38.9%	39.6%	41.6%
CHO	1765	1609	1657
	47.2%	49.1%	47.3%
Total Cals	3850	3363	3663

Table 13. Demonstrating differences in energy requirements in four differing working groups at Halley Bay.

The body weights are shown for three specific months during the year with maxima and minima. Also demonstrates the mean basal requirement (calculated from nomograms) and the mean total calorie intake. In column 11, the calorie intake has been presented per kg body weight, and in column 12, the excess of calories over the basal requirement has been shown as a percentage of the basal.

Group	No.	No. of assessments of dietary intake	Height (cm)	Body Weight Feb '61 (kg)	Body Weight July '61 (kg)	Body Weight Jan '62 (kg)	Maximum Body Wt. (kg)	Minimum Body Wt. (kg)	Basal requirement	Total Cal. intake	Cal intake/ kg body wt.	Excess cal. intake over basal as percentage of basal
Manual Workers	6	48	182.6	78.1	79.8	80.6	82.0	77.7	1953	4115.3	51.6	109.0
Scientists	9	72	180.3	77.1	80.1	79.8	81.8	76.7	1947	3729.4	46.6	91.6
Technicians	8	64	176.9	70.1	73.6	72.8	74.6	70.0	1841	3152.6	42.8	71.8
Cooks	2	16	178.8	68.6	70.4	72.8	73.0	68.6	1845	3227.9	45.9	74.9

the total calories at the beginning of the year, and for the percentage of calories furnished by carbohydrate to show a slight elevation in periods III and IV, the percentage compositions of the diets remained remarkably constant. In Table 12 it may be seen that the fat percentage showed a very slight tendency to rise from the first season to the third season. Clearly, these changes are minimal and would not stand up to a statistical examination.

In Table 13 the calorie intakes for four working groups in Antarctica have been tabulated, namely: manual workers (subjects ED, DED, RL, JS, GT and CJ), technicians (MBR, DJ, EF, PN, AP, MS, MT and ET), scientists (DA, MB, GB, CD, DE, MJ, SM, MTH and BP), and cooks (AM and GM). Also in the table are shown the relevant data on body weight changes; the excess calorie intake over the basal, expressed as a percentage of the basal; and the calorie intake/kg body weight. Although, numerically, the mean values of the food intake with the ratios of the excess calorie intake to basal metabolic rate and the ratio of the total calorie intake to body weight showed differences, the only significant difference was between the calorie intake of the manual workers and that of the technicians ($p \leq 0.02$).

Table 14 shows the mean percentage compositions of the calorie intakes of the four occupational groups. The differences between the groups can be seen to be minimal and without doubt are non-significant. In table 15 the caloric requirements have been computed for a standard reference man of 70 kg, and it is seen that all levels are quantitatively reduced.

3. Discussion

a) Body Weight

Although the findings of other expeditions who have investigated

Table 14. The mean percentage composition of dietary intakes for four working groups at Halley Bay.

Occupation	Number	Protein	Fat	CHO
Manual Workers	6	11.9	39.7	48.4
Technicians	8	11.7	39.6	48.7
Scientists	9	12.2	40.4	47.4
Cooks	2	12.2	38.8	49.1

body weight changes in cold regions do not all tally, it is now almost universally accepted that at the beginning of the year there is a weight gain. During the first two months of the present experiment there was a mean weight gain of 2.5 kg. The mean weight increased a further 0.6 kg by August and September, and then fell by 0.4 kg from September to January. It is clear that the smaller changes after the initial increase are non-significant trends. Lewis, Masterton and Rosbenbaum (1960) and Wilson (1960) observed an interseasonal effect upon body weight. They noted a depression in weights on the occurrence of the warmer summer temperatures after those of the winter. Lewis et al point out that the conspicuous weight rise at the beginning of the year was well in advance of the low temperatures of the winter period. They also suggest that the winter weight gain results from reduced physical activity rather than increased food intake. However, these findings are countered by those of Goldsmith (1959), who found that the body weights of members of the Trans-Antarctic Advance Party fell during the winter. In this case, it is probable that the men were in negative calorie balance; they were on reduced rations, and the energy expenditure remained at a high level. Sparke (personal communication, 1963) observed a 5 kg rise in body weight in the period spent in the ship between the United Kingdom and Antarctica, and this was followed by a sustained rise in mean body weights through the following year. There was no marked rise during the winter.

It is true that in the latter months of the winter at Halley Bay, the subjects demonstrated their maximum body weight. However, the fall off at the end of the year was small, and does not compare in amplitude with that found by Lewis, Masterton and Rosenbaum (1960) and Wilson (1960). It can only be concluded that the absence of a specific pattern in the body weight changes of expedition personnel is explained by an interplay

Table 15. Calorie requirements for differing working groups in Antarctica, for a standard reference man of 70 kg.

Occupation		Calorie requirements
Manual Workers		3612
Technicians		2996
Scientists		3262
Cooks		3213

of agents which act either to increase or to decrease this parameter. These factors to a large extent are environmental, but it is clear that they exert varying effects. Crudely, it may be said that there is a triangular relationship between the three factors: the climate, the energy balance and the psychological stress.

Whether the increase of weight at the beginning of the year is entirely due to a positive calorie balance has yet to be explained; more clearly, can the increase be instigated by the organism in order to augment its resistance to cold stress ? At Halley Bay, the greatest weight increase occurred when the energy expenditure was at its highest level, i.e. during the time of the building programme. From the shape of the curve (Figure 5) the preliminary gain in body weight would fit in with a hypothesis that it was secondary to the cold stress and that it might be a change accreditable to acclimatization. In the following pages, evidence from the present work will be discussed which helps to clarify the issue.

Martin (1949) reported from a survey of more than 91,000 recruits to the services that the average weight was 61.7 kg, and the average height was 171.5 cm. The mean body weight of the Halley Bay subjects was 74.3 kg at the beginning of the experiment, and the mean height was 179.6 cm. Thus they were heavier and taller than an average group in the United Kingdom. Lewis, Masterton and Rosenbaum (1960) and Orr (1962) and many others have reported a tendency for expedition personnel to be of greater height and weight than the average.

By the time the body weights of the subjects had reached constancy at a level of 77.0 kg, using the means for height and age as previously stated, from the tables drawn up by the Actuarial Society of America (Medical Impairment Study, 1912) the subjects were found to be 9.4 kg overweight.

b) Skinfold Thickness

The mean relative body weight of the group was found to be 113.7% and above the average for subjects of mean age 24 years and mean height 179.6 cm. Brozek and Keys (1951) measured the body weights and skinfold thickness of a group of students of college age. For purposes of classification of the subjects into categories of fatness, the authors used the twentieth, fortieth, sixtieth and eightieth percentiles. These 'quintiles' divide the distribution curve numerically into five equal groups of individuals. The mean relative body weight of the Halley Bay group was equivalent to that on the eightieth percentile of the group studied by Brozek and Keys. However the mean skinfold thickness readings were: abdominal < 20th; pectoral <20th; scapula< 20th; lateral arm < 40th; lateral thigh = 60th percentile. Thus though the relative weight of the Halley Bay group was well to the right of the distribution curve obtained by Brozek and Keys, the skinfold measurements were distinctly inclined to the left of the curve. The conclusion is that the Halley Bay subjects were more muscular than the average American college student, i.e. that their excess weight was not due to adipose fat.

Welham and Behnke (1942) in a study of professional football players demonstrated that 'overweight' cannot be simply identified with obesity. The football players were actually thin in the sense of having a low fat content in the body, although the average body weight was 24.6% above the army standard for men of the same age and height. It is unfortunate that there are no available figures for a population of young male adults in Britain, for the comparison of the skinfold thickness measurements.

From Figure 6 it will be seen that there was an increase in the skinfold thickness at the scapula, lateral arm, abdominal and pectoral

sites, from the initial reading in February to the reading in May. For the remaining months the readings showed little fluctuation, and stayed at a constant level. The salient feature of this portion of the experiment was the increase which occurred between April and May. It has been stressed that the mean body weight had levelled off by April. Thus there was an increase in the skinfold thickness without a concommitant increase in body weight. Lewis, Masterton and Rosenbaum (1960) established a good correlation between changes in skinfold thickness and body weight. However, a closer examination of their results reveals that the respective maxima and minima do not coincide on the time scale. It is noteworthy that the increase in skinfold thickness between April and May at Halley Bay was well marked. To suggest a reason for this anomalous finding from such scant data is impossible. The lack of correlation between the skinfold thickness and body weight at the beginning of the year can be shown in a more dramatic manner.

Formulae for the prediction of total body fat from measurements of the skinfold thickness at three sites (triceps, scapula, and abdominal) have been propounded by Edwards and Whyte (1962) who report that the most satisfactory is that based on skinfolds x height^2, viz.

$$\text{Fat mass} = 0.11\ (\text{S.F.} \times H^2 \times 10^{-4}) + 3.1$$

In the latter formula, S.F. denotes the sum of the three skinfold thickness measurements which were made. In the case of the present project, the triceps reading was not recorded but the measurement made on the lateral side of the upper arm was used in its place.

In the months February, April and May the estimates of the total body fat for the 'average base man' of 179.6 cm in mean height were 12.4, 13.4 and 14.4 kg respectively. Over the same period the mean body weights of the group was in February 74.3 kg, in April 76.8 kg and

in May the mean body weight was 76.6 kg. Thus the marked discrepancy is again seen between the assessments of the actual weight increases and those made from predictions based upon the skinfold thickness readings.

The April to May increment in skinfold can be accounted for by three suggestions. It is apparent that during a period of change in body weight, the correlation between the latter and between skinfold thickness changes is not close; and also it is seen that the skinfold thickness may increase without a parallel increase in weight. Thus it may indicate changes in fluid compartments are occurring. It is generally agreed that plasma volume decreases after a period of exercise and the increase in skinfold which occurred in the third month may have represented a shift in fluid compartments as a result of the reduced physical activity which occurred at this time. The shift would have occurred from the plasma into the inter- or intra-cellular compartments. Secondly the April to May skinfold thickness increment was coupled with a very slight fall in the monthly mean body weight and this may indicate that there was a metabolic exchange of glycogen stores into fat, with associated loss of body fluid. That part of the weight increase at the beginning of the year may have been the result to some extent of a water retention associated with storage of carbohydrate (vide infra) fits in with this view. Thirdly, that the phenomenon may have been due to a mobilization from the fat depots in other regions of the body, cannot be denied or substantiated, but the suggestion may be discarded by reason of its improbability.

Orr (1962) also observed a well marked dissociation between the skinfold thickness and the body weight. He showed that at the beginning of the Antarctic year the subjects at Hope Bay (Graham Land) demonstrated a sharp drop in skinfold thickness whilst the body weight remained at a constant level. Orr concludes that the manifestation resulted from a

fitness change, the men laying down muscle tissue and losing fat, the weight thereby remaining constant. He also noted no seasonal change in body weight; the base was further North than those in which seasonal changes are usually noted and also, sledging was the main form of occupation through the year. Sparke (1963) noticed similar dissociations between skinfold thickness and weight changes in an Antarctic group.

No real conclusion can be gathered from the skinfold thickness measurements; although they tend to follow rises or falls in body weights the correlation is not so definite as has been previously thought. The whole subject demands further research; by no means the least of the problems is the question: what is it that is being measured with the calliper? Clearly, there is more than one variable; besides changes in subcutaneous fat thickness, the fluid content of the organism must be a factor. Differences in population groups or individuals also may be more pronounced than the absolute thickness of the subcutical fatty layer would portend from autopsy studies and such differences may be to some extent explained by laxity or tightness of the skin, or by the amount of superficial fascial tissue. The survey poses problems to be answered; the present data is too scant to allow more than the most flimsy of conjectures as to the causes of these anomalous results.

c) Dietary Intake

i. Total Calories

From Table 9 it will be seen that the grand mean of 200 assessments of the food intake at Halley Bay amounted to 3597 kcal/man/day, and that the percentage of calories supplied by protein, fat and carbohydrate was respectively 12.1, 39.8 and 48.1%. From the standard deviation of the total calorie intake it can be calculated that 95% of the intakes lay between 1776 and 5418 kcal.

It has been suggested by many authors that the caloric requirements for cold regions would be of the order of 5500 to 6000 kcal/man/day, both for men engaged in field operations and for those on routine base duties (Johnson, Crowley, Toth, Koehn, Monahan, Lalanne and Parrott, 1949; Swain, Toth, Consolazio, Fitzpatrick, Allen and Koehn, 1949). Marked increase in calorie intake has been reported in the papers by Johnson and Kark (1947) and Kark, Croome, Cowthorpe, Bell, Bryans, Macbeth, Johnson, Consolazio, Poulin, Taylor and Cogswell (1948). However, a survey carried out among European trappers in Greenland (1939 to 1940) by Rodahl (1949) showed that the white trappers maintained their body weight on an average gross consumption per man of 3000 kcal/day. Also miners in Spitzbergen who worked a 9 hour day gained weight on an average consumption of 4500 kcal/man/day (Abs, 1929). Masterton, Lewis and Widdowson (1957) assessed the level for four men taking part in the British North Greenland Expedition (1952 to 1954) as being 3911 kcal/man/day while living on the base, and an intake of 4770 kcal/man/day for the same group while participating in a sledging expedition. Davies (1962) estimated the intake of one man living a routine base life in Grahamland to be 3295 kcal/day, while Orr (1962) pointed out that a per capita intake of 5000 kcal/day formed an excellent basis for a sledging ration. The energy requirements of adult Eskimos in Greenland of average weight 65 kg have been assessed to be 2000 to 2900 kcal/man/day from data obtained by Krogh and Høygaard (Krogh and Krogh, 1913; Høygaard, 1941).

It has also been pointed out in the case of United States soldiers in an Arctic environment that the percentage of calories furnished by protein, fat and carbohydrate are not significantly different from those reported for troops eating garrison rations in temperate climates (Johnson and Kark, 1946; Swain et al, 1949).

The above review indicates that the consumption of the Halley Bay subjects was on the low side when compared to the findings of the majority of workers. The average intake was higher than that observed by Rodahl (1954) in his studies in Infantry and Airforce groups stationed in Alaska living on separate bases, and following differing routine existences. The average intake for the Infantry group was 3200 kcal and that for the Airforce group was 2900 kcal/man/day. However the subjects of these groups were in isocaloric balance, but the Halley Bay personnel gained 2.5 kg in mean body weight in the first 2 months after which they maintained caloric balance. If the mean level for the second summer (Periods VI, VII and VIII) where the body weights were stable, is substituted for that of the first summer (Periods I and II) thus eliminating the excess calories which caused the weight increase, a figure of 3429 kcal/man/day is obtained, which remains higher than Rodahl's two groups, but agrees with Norman's result (1960) for mean energy expenditure of Halley Bay scientists.

It is of interest to compare the results of the Antarctic survey with some energy intakes recorded in the United Kingdom. The mean intake of the Halley Bay subjects agreed closely with levels reported for industrial workers (Bransby, 1954) intermediate Sandhurst cadets (Edholm, Fletcher, Widdowson and McCance, 1955) and army troops on exercise (Adam, Best, Edholm and Wolff, 1957). It is lower than the evaluation of 4030 kcal/man/day made for miners by Garry, Passmore, Warnock and Durnin (1955) and higher than the level of food intake for students, namely 2950 kcal/man/day, as estimated by Kitchen, Passmore, Pyke and Warnock (1949). The result of the Halley Bay survey corresponds with the intake that would be expected in a man performing moderately active work, such as a carpenter. Masterton, Lewis and Widdowson (1957) measured energy expenditure in four men while on the British North

Greenland Expedition and reported a mean level of 3581 kcal/man/day, which is remarkably close to the result of the present survey.

The mean percentages of protein, fat and carbohydrate for the year show little difference when compared with the composition of food intake stated in the Annual Report of the National Food Intake Committee (Ministry of Agriculture, Fisheries and Food, 1962) for domestic food consumption for 1960, namely 11.5, 39.3 and 49.3% respectively for the three food groups. From the industrial survey of Bransby (1954) the author calculated the protein, fat and carbohydrate composition as being 13.3, 37.8 and 48.9% of the total calories. In a dietary survey involving ninetynine bank men in the United Kingdom, Morris, Marr, Heady, Mills and Pilkington (1963) assessed the percentage of calories supplied by fat to amount to 41% which is above the Halley Bay result. It does not appear that any particular food group was consumed in excess over the intakes recorded in temperate climates.

From the weighted means shown in Table 12 it can be seen that the mean for the first summer season was 187 kcal higher than that of the second summer. With the assumption that the energy output of the two summers were approximately equal, these excess calories may be said to have caused the increase in weight which occurred in the first two months. The results of the first two dietary periods overlap the two months during which the subjects showed the weight increase, but if the median point in time is taken for the second survey session, which would be 9 weeks after the initiation of the experiment, the discrepancy is not so great.

The total excess of calories which caused the weight increase would amount to 10472 kcal taking a time of 8 weeks for the excess intake. From this figure it may be concluded that 4188 kcal were required to

increase the mean weight by 1 kg. This figure shows a discrepancy when compared with the result of Keys, Anderson and Brozek (1955) who found in a study of individuals who had not previously fasted that on a high calorie diet the calorie equivalent of 1 kg of tissue mass gained was 6180 kcal, while the figure of Strang, McCluggage and Evans (1930) of 7700 kcal/kg body weight increase shows a greater difference. The calorie equivalent of body weight is dependent on the state of the protein and carbohydrate stores in the body. It might be assumed therefore using the results of the experiment, in which the calorie intakes may not show the true picture, that the weight increase was not only due to the laying down of fat stores in the depots. The question arises that, after the period of inactivity which the subjects underwent while on the supply ship, and during which their protein resources may have been catabolised, on reaching the base with the dramatic increase in activity which then ensued, was their weight increase partially due to the laying down of protein, combined with the storage of depot fat.

Leathes and Raper (1931) pointed out that whereas fat can be stored in the almost pure state, carbohydrate or protein cannot be stored dry but will retain with them three or more parts of water. It may be concluded that a portion of the weight increase was due to the intake and laying down of stores of protein or carbohydrate, with the concomitant retention of water. It is interesting to note that the fat intakes of the first and second summers are almost equal and that the extra calories were provided by the protein and carbohydrate. That energy expenditure during the first season was less than that of the last is doubtful, the entire manpower being employed in full scale building operations during this part of the year; in fact the tendency would have been for greater energy expenditure.

The problem of the causation of the weight gain may be approached from another venue. Using Strang et al's figure for the caloric equivalent of weight gain, for an increase of 2.5 kg 19250 kcal would be required. Over a period of 8 weeks this would mean that there would be an excess of 344 kcal/man/day. Thus for the first season, the estimated intake would have to be reduced by 344 kcal to 3506 kcal/man/day to give the true energy requirement. This result is low in comparison to the figure for the second summer (3663 kcal) at which time the weights were stable, and appears improbable. The average summer mean for energy requirement would be 3584 kcal/man/day by employing the above results, which is lower than that estimated for energy expenditure for scientists in the Antarctic by Norman (1960). This again lends support to the hypothesis that the gain in weight is not entirely due to the deposition of fat in the adipose organs.

However, the fact remains that the skinfold thickness increased over the first 3 months, which would suggest a possible storage of fat, though this has been queried in the discussion concerning these parameters. The suggestions made from the dietary survey in connection with changes in body weights are therefore in opposition to those made from the skinfold thickness results. It is a paradox which cannot be explained.

The depression of the calorie intake during the winter is not entirely unexpected as the energy expenditure was considerably reduced as a result of the period of darkness and low outside temperatures. The continuation of the reduced intakes beyond the end of the darkness is again due to the lack of exposure and energy expenditure caused by the prolongation of the low temperatures. From the first to the second seasons there was a fall of 486 kcal (12.6%) in total calorie intake, and a rise of 299 kcal(8.2%) from the second to the third season. The winter drop in calorie intake of the Halley Bay subjects shows agreement

with studies by Rodahl on trappers in the Arctic (1949), in whom the summer mean was 3300 kcal/man/day, and the winter mean was 2000 kcal, the latter intake being to some extent a result of a self-imposed rationing scheme . Milan and Rodahl (1961) found the opposite effect in a dietary survey of personnel in Little America V, who gained weight markedly on an increased calorie intake in the winter months. Durrer and Hannon (1962) measured the food intake of dogs in an Arctic environment and showed that there was a marked winter rise in total calorie intake. They also showed that the body weights were higher in the summer than in the winter months. The above findings are in direct contrast to the Halley Bay results.

Norman (1960) in Halley Bay scientists evaluated the mean energy expenditure for the year as 3387 kcal/man/day but his winter figure is largely responsible for this lowish result (3120 kcal) and his summer figures show a closer correspondence with those of the diet survey. His depression of 651 kcal from his summer mean of 3771 kcal is of greater magnitude than that shown in the survey, but his group was smaller, and was composed of those following scientific projects and did not take into account the manual workers. However, the fact that an undoubtedly significant drop in energy expenditure was obtained during the winter justifies the results of the nutritional survey. The belief of Lewis, Masterton and Rosenbaum (1960), that the winter increase in weight they observed was due to a falling off of activity and not of calorie intake, is not substantiated.

ii. Protein

The seasonal effect in the protein intakes was found to be highly significant at the 0.1% level. The drop after the first two periods was marked, amounting to 148 kcal (28.4%) and was followed by a rise of

only 4.3% from the second to the third seasons. The calories of the lowered protein intake were taken over during the winter by carbohydrate in preference to fat, but in the third season the fat was elevated to replace the protein calories. The maximum mean protein value for a survey period was one of 528 kcal (14.2% of total calories) which occurred in the first session, and the minimum one of 362 kcal (11.1% occurred in the fourth period in the depths of the winter. The lowest calorie protein intake when expressed as a percentage of the total became apparent during the sixth period (10.3%) and is below the minimum protein allowance proposed by the British Medical Association's Committee on Nutrition (1950) who considered that protein should contribute 11% of the total calorie intake. However the League of Nations Commission on Nutrition (1936) suggested that protein intake should not fall below 1 gm/kg body weight and the above figure is well within these limits. It must be stressed that at no time was the protein source scarce or rationed and that these results represent the subject's natural inclination for protein, although it remains that the men often expressed desire for more fresh meat in preference to the tinned meats which were the stable supply.

The lack of significance in the 'between men' effect in the statistical analysis coupled with the very significant effects in the carbohydrate and fat intakes, may indicate that the protein consumption is dependent on the presentation and cooking of the food, whereas the fat and carbohydrate rely more on variables such as body weight and occupation.

iii. Fat

The period changes in the calorie fat intakes are demonstrated in Figure 7 and the values are tabulated in Tables 10 and 11. Analysis

of variance showed that the depression in the winter readings was highly significant at the 0.1% level both for the 'between seasons' effect and the 'between men' effect. The depression of 169 kcal from the first season to the winter was approximately equivalent to the rise which occurred during the second summer. It seems likely therefore that the changes in fat intake parallel the energy expenditure results of Norman (1960) for the elevation in the protein and carbohydrate levels were not nearly so great at the end of the winter. The fact that the 'first' and 'second summer' calorie fat intakes were so similar lends support to the hypothesis made previously that much of the increase in weight at the beginning of the year was due to the laying up of stores of protein and carbohydrate, the fat calories being primarily for energy requirements. From Figure 7 it may be seen that the fat calories showed a tendency to mimic the pattern of the total calorie changes to a greater extent than either the protein or the carbohydrate calories.

The means of the fifth dietary period are of interest since it is at this stage that the period of darkness is drawing to a close but the outside temperatures are at their minimum; however the fat calories showed a marked rise, although the carbohydrate calories fell to their lowest level, and it may be that this elevation of the fat calories reflects a slightly increased exposure to cold which could well have occurred at this time. It is only at this point that the fat calories do not correlate with the total calories.

iv. Carbohydrate

The carbohydrate consumption showed the least significant changes in the analysis of variance for a seasonal effect but a 'between men' effect was highly significant. The winter intake of carbohydrate was 165 kcal lower than the ' first summer' intake of 1765 kcal and the

'second summer' intake was only 48 kcal higher than that of the winter. The winter figure as a percentage of total calories was almost 2% higher than either of the summer intakes and there appeared to be a slight tendancy to consume a higher carbohydrate diet in the winter months. From Figure 7 it can be seen that the carbohydrate values showed an inverse relationship to the fat intakes, which is the natural result of the protein intake maintaining a reasonably constant level. The elevated carbohydrate intake in the first two dietary sessions over that in the last three, with the associated rise in body weight, and the parallel rise in skinfold thickness measurements (see Figure 6), may indicate that some of the carbohydrate was being metabolised into adipose tissue.

d) The Energy Requirements of Four Occupational Groups

The mean total caloric intakes of the four occupational groups, demonstrated in Table 13, indicates that the manual workers consumed a diet approximately equal to that eaten by miners. However, when the calorie requirements are scaled for a standard reference man of 70 kg body weight (the average weight of the group of manual workers was 78.1 kg) the dietary intake fell to 3612 kcal/man/day, which is equivalent to that of a moderately active worker. The technicians showed the lowest mean food intake at 3152.6 kcal/man/day, which for the reference man was calculated to be 2996 kcal/man/day. The intake of the technicians was therefore equivalent to the intake of various groups of students (Kitchen, Passmore, Pyke and Warnock, 1949; Cook and Wilson, 1962) and of a group of 10 clerks assessed by Garry, Passmore, Warnock and Durnin (1955). The intake of the scientists was intermediate between manual workers and technicians and was similar to that of the intermediate cadets (Edholm et al, 1955) or that of industrial workers

(Bransby, 1954).

From the data concerning the change in body weights it will be seen that the manual workers showed the lowest maximum gain (3.9 kg), while the scientists showed the highest maximum gain (4.5 kg). However, the difference is small, the gains to all intents and purposes being equal. From Table 13 it is seen that the ratios in the last two columns (Calorie intake/kg body weight, and Excess calories over the basal expressed as a percentage of the basal) bear the same relationship to the occupational groups as the total caloric intakes.

Though the intakes of the scientists and technicians were not significantly different, the disparity is such that comment cannot be avoided. It is difficult to explain why the food consumption of the scientists should be so much higher than that of the technicians. For many of the scientists the work was largely sedentary, but it may be that they worked for longer hours than the technicians, or that their motivation for completing their work was of greater magnitude. From personal observation at Halley Bay, differences of such magnitude in energy expenditure were by no means obvious.

The results of a survey of dietary intakes and occupations by Mayer, Roy and Mitra (1956) on two hundred and thirteen mill workers in West Bengal may be relevant to the present study. The authors reported that caloric intake increased with activity only within a certain zone ('normal activity'). Below that range ('sedentary zone') a decrease in activity was not followed by a decrease in food intake but by an increase. Thus the energy expenditure of the Halley Bay scientists might have been lower than that of the technicians even though they were consuming more calories. However, if this were the case, there surely would have been a greater increase of body weight in the scientists than in the technicians

and from Table 13 it is seen that the gains shown in body weights were virtually equal.

e) General Discussion

It has been stated that any process of acclimatization would present either as a comparatively rapid increment or decrement in a parameter under investigation in the first few weeks of the year, or as a continuous rise or fall throughout the year. The changes in body weight and skinfold thickness would fit in with such a premise. The fact that the calorie intake at the end of the year was less than that of the first season suggests an overall downwards trend. The possibility arises therefore that the high food intake, the gain in body weight and the increase in skinfold thickness may have been a response due to acclimatization. Such a hypothesis cannot be eliminated. The fact that the energy expenditure at this time was at a maximum level makes it seem more probable that the change was due to cold stress. The reason for the disparity in the results of various expeditions can only be explained by the fact that the variables of cold, energy expenditure, food intake, and possibly of psychological stress each play leading or subservient roles at different times.

4. Conclusions

i. That there was an increase of 2.5 kg in body weight during the first 2 months of the survey, after which the changes were small. There was only a very slight fall in the last months of the year. The gain in body weight occurred well before the winter period of reduced temperatures and polar night.

ii. The results of the skinfold thickness measurements were highly

significant for a 'between seasons' effect and a 'between men' effect when examined by variance analysis for the readings taken from the pectoral, scapula, lateral arm and the abdominal sites. The results showed that the mean monthly values of the four sites mentioned increased from February to April, thus showing some dissociation from the body weight changes. The anomaly, however, cannot be explained. Some conjectures have been made as to the causes of this dissociation.

iii. The mean total calorie intake was 3597 kcal/man/day from 200 assessments employing a population of twentyfive young men. The intake is similar to that recorded in the United Kingdom for industrial workers and intermediate Sandhurst cadets. The above figure shows reasonably close agreement with the energy expenditures of Halley Bay scientists obtained by Norman (1960). Thus there seems to be little evidence that men on 'static' bases increase their food intake as a result of cold stress.

iv. The percentage composition of the dietary intake did not markedly differ from that of the national average.

v. Using the analysis of variance, significant 'between season' effects were established for total calories and protein, fat and carbohydrate calories and significant 'between men' effects for total calories and fat and carbohydrate calories. For total calories, and fat calories, the winter intake was found to be lower than that of either the first or the second summer seasons. This was also true for the protein and carbohydrate calories but they were regained in the second summer to a far lesser extent than the total calories and the fat calories. A rise in fat intake was largely responsible for the rise in total calories

in the second summer season.

vi. That the weight gain recorded in the first 2 months of the year was not entirely due to the laying down of fat stores, but also could be accredited to the storage of protein and/or carbohydrate.

vii. That the average energy requirements of the four occupational groups, namely manual workers, scientists, technicians, and cooks, were respectively 4115.3, 3729.4, 3152.6 and 3227.9 kcal/man/day.

viii. It cannot definitely be inferred that any of the monthly changes in body weight, skinfold thickness and dietary intake result from exposure to cold, or indicate a process of acclimatization to cold stress, however the results are highly suggestive that this is the case.

SECTION V. VARIATIONS IN BLOOD PRESSURE AND PULSE RATE

1. Experimental Method
2. Results
3. Discussion
4. Conclusions

Table 16 . The mean monthly value, with the standard deviations of systolic and diastolic blood pressure (mm Hg) and of pulse rates (beats/min). Also shown, the pulse pressure.

		Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sep	Oct	Nov	Dec	Jan 1962
Systolic	Mean	108.8	107.3	109.1	106.3	105.0	106.1	108.1	112.0	109.3	115.9	109.7	111.8
	SD	13.1	10.1	9.9	13.4	13.1	15.5	13.6	9.4	12.2	12.0	14.1	15.0
Diastolic	Mean	69.4	72.7	72.5	73.3	72.4	70.5	74.8	75.9	73.3	77.3	71.5	72.5
	SD	9.5	9.4	9.6	11.1	10.7	9.0	9.6	8.8	11.1	10.3	8.5	12.4
Pulse pressure		39.4	34.6	36.6	33.0	32.6	35.6	33.3	36.1	36.0	38.6	38.2	39.3
Pulse rate	Mean	57.7	58.3	58.1	59.2	57.9	56.0	57.0	58.3	56.4	58.1	56.5	56.2
	SD	6.1	6.5	9.4	7.1	7.0	4.9	7.1	7.2	4.7	7.7	5.7	8.1

1. Experimental Method

The blood pressure and pulse rate of twentythree men were estimated at monthly intervals. The assessments were made before the subjects arose in the early morning, on the same days of the venepunctures and adipose biopsies. The systolic and diastolic pressures were measured twice indirectly with a mercury sphygmomanometer according to the method outlined by Bramwell (1940) and the pulse rates were evaluated over a period of one minute. It is stressed that these measurements were carried out under absolute resting conditions, and can therefore be termed sleeping levels. All measurements were made by the author.

2. Results

The monthly averages of the systolic and diastolic blood pressure, the pulse rates and the pulse pressure are shown in Table 16. It is seen that there was no marked variation in any of these parameters. There was a very slight tendency for the systolic blood pressure to rise towards the end of the year and the trend was shown to a lesser extent in the diastolic pressure. In the months May, June and July the systolic pressure was at its minimal level and this was reflected in the pulse pressure for these months. The pulse pressure showed some evidence of a seasonal effect, falling from 39.4 mm Hg in February 1961 to 32.6 mm Hg in June, and rising to 39.3 mm Hg in January 1962. The pulse rate remained at a constant level throughout the year.

To test the statistical significance of the very small monthly changes in systolic blood pressure and pulse pressure would be irrational, in view of the methods by which the recordings were made. Readings performed with the spyhgmomanometer may have clinical significance when

Table 17 . The mean values for the systolic and diastolic blood pressure (mm Hg), the pulse pressure, and the pulse rate (beats/min) for the first and the second halves of the year.

	1st Six Months	2nd Six Months
Systolic BP	107.1	111.1
Diastolic BP	71.8	74.2
Pulse pressure	35.3	36.9
Pulse rate	57.9	57.1

demonstrating large deviations from the norm, but for the very slight changes shown in these parameters in the present survey, even if significance were established, it is possible that the perception of the observer might have been at fault and have accounted for the seasonal changes. Table 17 shows that the mean values of the blood pressure, pulse pressure and pulse rate were virtually the same for the first 6 months compared with the second 6 months.

3. Discussion

The survey of the blood pressure and pulse rates was carried out mainly as a comparative parameter to the serum lipid levels which will be discussed in Section VI, particularly in connection with the individual mean values for the year.

During preliminary discussions with workers at Hampstead, the fact that severe hypertension has become manifest in a small proportion of men taking part in polar expeditions was raised. L.G.E. Pugh (personal communication, 1960) has claimed that cases have occurred in great enough numbers to make the subject worthy of investigation. It was reported at a symposium on polar medicine (Lancet, 1959) that a casual blood pressure reading made on a member of the Trans-Antarctic Expedition at the South Pole showed severe hypertension. A measurement made some weeks later indicated that the blood pressure had returned to normal. It is also relevant that two papers have reported hypertension in sub-Arctic labour camps (Hohorst, 1957; Ott, 1957). In the present survey, there were no cases of hypertension, in fact the opposite of this tended to be the case.

The interesting finding of the survey was that the overall mean value of the blood pressure was on the low side for a group of

men of average age twentyfive years. In the R.A.F. the lower level of normal has been accepted as being a blood pressure of 110/70 mm Hg (Rook and Dawson, 1938) and the Halley Bay result appears to be remarkably close to this standard. Tables 33, 35 and 36 indicate that out of the group of twentythree subjects, thirteen had a systolic pressure lower than 110 mm Hg, and 6 had a diastolic pressure lower that 70 mm Hg. The comparatively low blood pressure agrees with the mean level reported by Davies (1962) in a survey of a small group of Antarctic personnel in that the diastolic pressure was just below 70 mm Hg and the systolic pressure was just above 110 mm Hg. From these results it is concluded that life on a static Antarctic base has no adverse effect on the blood pressure of young adults. The resting blood pressures and pulse rates taken after expeditions into the field again demonstrated no elevation over the base values.

The slight seasonal changes in systolic blood pressure and pulse pressure, though clearly non-significant, do compare with a serial survey of blood pressures made by Tikhomirov (1963) on men domiciled in the inland regions of Antarctica. During the winter months, the pulse pressure fell below the summer values; the author, however, made no comment in connection with these changes. The men on the inland base were subjected to very similar conditions as the Halley Bay members, with the exception that the Russian station was situated at approximately 10,000 ft above sea level. To hypothesise on such scant data as to the causes of this seasonal change is questionable; it can merely be stated that the pattern fits into the changes which occur in physical activity and outside exposure, which are both greatly reduced in the winter months, i.e. during the reduced physical activity systolic pressure and pulse pressure were lower than when activity was high.

MacLean (1919) investigated changes in blood pressure of men taking part in the 1911-1914 Australasian Antarctic Expedition. He reported that there were no distinct seasonal changes and showed that in the winter the blood pressure rose, which continued into the summer months. MacLean attributed some of the rise in systolic pressure to increments in energy expenditure. Davies (1962) demonstrated no seasonal effect in a survey of blood pressure and pulse rate in Antarctica.

4. Conclusions

The conclusions from this part of the work are therefore that there was no evidence that hypertension occurs in Antarctic personnel as a result of cold exposure, and that a seasonal change was present in pulse pressure and to a lesser degree in systolic blood pressure, which was small in amplitude and it was clear that it was statistically non-significant.

SECTION VI. VARIATIONS IN SERUM LIPID CONCENTRATION

A. EXPERIMENTAL METHODS

- a) In the Field.
- b) In the Laboratory
  - i. Triglycerides
  - ii. Cholesterol
  - iii. Phospholipid

B. VARIATIONS BETWEEN SEASONS

1. Total Lipids and Total Fatty Acids
   - a) Results
   - b) Comments
2. Phospholipids, Total and Free Cholesterol, Cholesterol Esters and the C:P Ratio
   - a) Results
   - b) Comments
3. Alpha-and Beta-cholesterol
   - a) Results
   - b) Comments
4. The Influence of Sledging on Serum Lipids
   - a) Background
   - b) Results
   - c) Comments
5. Discussion on Alpha- and Beta-cholesterol variations
6. Triglycerides
   - a) Results
   - b) Comments
7. General Discussion

C. VARIATIONS BETWEEN MEN

1. Results
2. Discussion
3. Conclusions

Monthly samples of serum were taken and stored in deep freeze in Antarctica. The serum was analysed for the following lipid levels in the laboratory; total lipids, total esters, triglycerides, phospholipids, total cholesterol, cholesterol esters and beta-and alpha-cholesterol.

A. EXPERIMENTAL METHODS

a) In the Field

Twentyfour members of the expedition agreed to give monthly blood samples. An average of six samples were taken daily, starting on the 10th of each month. The blood was drawn after an overnight fast. The cooks were asked to provide an evening meal of low fat content, i.e. one which would avoid fried and roast foods. At the same time subjects were requested not to take snacks high in fat, as was their custom before retiring for the night. As the venepunctures were carried out about 10 hours after the last major meal, the blood specimens cannot be said to have been absolute fasting samples, but, nevertheless, are comparable with each other in that the fast was over a constant period of time.

The samples were taken while the subjects were in their bunks before they had risen for their breakfast. Having noted their blood pressure and pulse rate the sphygmomanometer cuff was retained in place and the pressure raised to 90 mm Hg. A small area over the anterior cubital vein was cleaned with absolute alcohol, and was anaesthetised with 2% Xylocaine. A sterile transfusion needle attached to a six inch length of polythene tubing was inserted into the vein, and the blood was allowed to run into a 50 mg centrifuge tube. When 50 ml of blood had been taken, the pressure of the sphygmomanometer cuff was released and the

flow of blood was stemmed with a cottonwool swab. The puncture wound was covered with a protective elastoplast dressing.

The blood samples were then allowed to clot at room temperature (approximately 20°C) over an 8-hour period, after which the specimen was spun on a B.T.L. "Bara" Byro Centrifuge (Baird and Tatlock) at 3500 revs/minute. The supernatant serum was pipetted off with sterile Pasteur pipettes and placedin 6 oz medical flats for storage. The bottles which had been previously sterilised contained preplaced antibiotic (5 mg procaine penicillin and 5 mg streptomycin) to prevent bacterial decomposition should deep freeze conditions be unavailable at a later date. One µl DL-alpha-tocopherol (Roche) was also added as an antioxidant; the oily vitamin E preparation was added to the empty bottles in petroleum ether and the latter was blown off. Twentyfive to 35 ml of serum were placed in the bottles, and pure nitrogen was blown over the samples, which were finally sealed and placed in deep freeze. The specimens were stored in an ice cave in the locality of the living hut. The temperature of the cave parallelled the outside temperatures at a lower level and did not at any time rise above -6°C. The samples always remained in the solid state.

The serum samples were transported to the United Kingdom in the deep freeze hold of the relief vessel, the Kista Dan, and were then transferred by ship and plane still under refrigeration, to the South African Institute for Medical Research, Johannesberg.

The response of the subjects to an operation which is regarded by many as distasteful, was surprising. Complaints against the venepunctures were rare, and it must be added that the careful use of local anaesthetic in a project which depends upon a voluntary population does much to allay any fears.

The changes in weights and skinfold thickness readings have already been discussed but it should be mentioned that these readings were made on the same day as the venepuncture.

b) In the Laboratory

The estimations of the blood lipid levels were all made in duplicate.

i. Triglycerides The neutral fats were estimated by two methods, the results of which were in good agreement. A modification of the method of Van Handell and Zilversmidt (1957) using activated silicic acid and isopropyl ether was used for the initial separation of serum triglycerides, cholesterol esters and free cholesterol from phospholipids, and the extracts obtained in this manner were hydrolysed by the method of Carlson and Wadstrom (1959). The phospholipid -free extracts were analysed for ester groups by a modification of the method of Stern and Shapiro (Antonis, 1960). Triglyceride values were calculated by subtraction of the cholesterol esters from that of the phospholipid-free ester group. Total lipid extracts were also examined for ester groups, and a triglyceride level was obtained by subtraction of phospholipid and cholesterol esters.

Reagents: All reagents and solvents used were Analar reagent grade. Isopropyl ether was freed from peroxides by passage through a column of activated alumina (heated overnight at 170°C) just before use.

1) Methanolic Hydroxylamine solution (2M) Solution A: 13.9 g hydroxylamine hydrochloride were added to 5 ml distilled water and 50 ml absolute methanol. This was washed until it had fully dissolved, and was made up to

100 ml with methanol.

2) Methanolic NaOH solution (3.5N), Solution B:
14 mg of NaOH were added to 5 ml distilled water and 50 ml methanol. This was warmed until dissolved and made up to 100 ml with methanol.

3) Alkaline hydroxylamine solution:
Equal parts of solutions A and B were mixed and allowed to stand for 15 minutes. The clear supernatant was filtered off just before use.

4) Stock ferric perchlorate solution:
1.0 gm of iron wire was dissolved in 125 ml 70 to 72% perchloric acid in a beaker by warming on a hot plate until the reaction began, and was then removed. When the solution was complete it was made up to 250 ml with distilled water and stored at $4^{\circ}C$. The solution was allowed to stand for 4 days before use.

5) Working ferric perchlorate solution:
6 ml of the stock was diluted to 120 ml with absolute ethanol just before use.

6) Standards:
A stock solution (0.7623 gm cholesterol acetate in 500 ml chloroform) was diluted in quantities of 10, 20, 30, 40 and 50 ml with 50 ml chloroform.

Procedure: One ml of serum was extracted with 25 ml Bloor's solution. A 3 ml aliquot of this was filtered and evaporated to dryness and put aside for total ester analysis.

For triglycerides, 4 gm of silicic acid were slurried with 5 ml iso-propyl-ether; 1 ml of serum was added dropwise with continuous

shaking. A further 20 ml of iso-propyl-ether and two glass beads were added. The tube was stoppered, shaken thoroughly, and then placed in a mechanical shaker for 30 minutes. The silicic acid was then allowed to settle and a 3 ml aliquot of the supernatant solution was evaporated to dryness. A standard was set up using 1 ml portions of the various dilutions, which were evaporated to dryness. A blank was also started at this stage. To all tubes containing blanks, standards, total ester groups and triglyceride esters were added 3 ml iso-propyl-ether and 1 ml alkaline hydroxylamine. The tubes were stoppered with plastic caps, shaken well and left in a 25°C water bath for 30 minutes. Six millilitres ferric perchlorate solution were then added, the tubes were shaken vigorously and kept in the dark for 30 minutes.

The colour densities of the solutions were read on an Evelyn colourimeter using a 515 mμ filter, the 6 ml aperture, and the bright light source with a fully polished colourimeter reflector. The instrument was initially set at 100% transmission with the blank.

Total lipids, total esters and triglycerides were calculated according to the following scheme:

Let total ester groups = X

Let triglycerides = Y

Phospholipids x 0.69 = A (fatty acids of phospholipids)

Cholesterol esters x 0.73 = B (fatty acids of cholesterol esters)

Hence Triglycerides = X - (A + B) or = Y - B

Theoretically X - (A + B) should = Y - B

The higher result was taken if there was a discrepancy, and multiplied by 1.044 to obtain pure triglyceride.

Total lipid = phospholipid + total cholesterol + B + pure triglyceride

Total fatty acid = A + B + Y (or X - (A + B) if higher)

ii. Cholesterol Free and total cholesterol were estimated by the method of Sperry and Webb (1950a). Free cholesterol was precipitated directly as the digitonide. The total cholesterol was first hydrolysed and then precipitated as the digitonide.

Free and Total Cholesterol

Reagents:

1) Solvents: acetone-absolute ethanol (1:1), acetone-ether (1:2) and ether. All solvents were Analar reagent grade.
2) Digitonin solution, 0.5%: 500 mg of digitonin were dissolved in 100 ml of 50% alcohol at 60°C.
3) Potassium hydroxide solution was made by dissolving 10 mg of pure KOH in 20 ml water. If a sediment developed the solution was filtered before use.
4) Acetic acid solution. Made by diluting 10 ml glacial acetic acid to 100 ml with distilled water.
5) Acetic anhydride: 99 to 100%.
6) Cholesterol solutions containing exactly 100 mg/100 ml glacial acetic acid. Working standards were made by suitable dilution of the stock solution with glacial acetic acid. The cholesterol was anhydrous and pure.

Procedure: One ml serum was added to 25 ml volumetric flask half filled with acetone alcohol. The sides of the flask were rinsed, it was then boiled, and finally brought up to volume at 25 ml. The contents were filtered using a Wattman's No.1 filter.

Two 5 ml aliquots were taken off for estimation of the total cholesterol and two for the free cholesterol analysis and then were placed in a 15 ml centrifuge tube. Five drops of potassium hydroxide were added to the total cholesterols. The aliquots were mixed with a glass rod and placed in a 37°C water bath for 30 minutes. Two drops of 1% phenolphthalein were added and the aliquots were acidified with 10% acetic acid. One ml of the digitonin solution was added and they were allowed to stand overnight. They were then centrifuged, the supernatant layer was decanted off and the remaining sediment washed with 5 ml acetone-ether. This was repeated but the precipitates were washed with 5 ml anaesthetic ether. Having been centrifuged and decanted a third time, they were placed in a 37°C water bath to dry. Two ml of glacial acetic acid were added, the tubes were placed in a boiling bath to dissolve and then were put into a 25°C water bath.

Two 5 ml aliquots of each diluted standard were measured into a centrifuge tube and five drops of potassium hydroxide were added and mixed, with the addition of two drops of phenolphthalein. This was acidified with acetic acid, and 1 ml of digitonin was added. A blank of 2 ml acetic acid was set up at this stage.

A mixture of 20 ml acetic anhydride and 1 ml concentrated sulphuric acid was then made. Four ml of this mixture was then added to all the tubes Thirty minutes later the solutions were read on an Evelyn photometer using the 660 mμ filter and a 6 ml aperture. The standards were equivalent to 100, 200 and 300 units on the Evelyn photometer, and total and free cholesterol were estimated from a graph constructed with the points.

Beta-cholesterol The beta-cholesterol was precipitated with calcium chloride in the presence of heparin by the method of Burstein and Samaille (1958) and the cholesterol in the precipitate was determined by the technique of Pearson, Stern and McGavack (1953).

The precipitation of the beta-cholesterol:-

Into a 15 ml centrifuge tube were placed 0.2 ml serum, 2.0 ml calcium chloride and 0.04 ml heparin. The tubes were left overnight and were then centrifuged. The supernatant fluid was decanted off, and the precipitate was drained by inverting the tube for 5 minutes. To the precipitate was added 0.2 ml of sodium citrate, and it was dissolved by stirring with a glass rod.

The determination of beta-cholesterol:-

Reagents:

1) Acetic anhydride.

2) p-Toluenesulphonic acid solution. In 100 ml of glacial acetic acid, 12 g of p-toluenesulphonic acid were dissolved.

3) Cholesterol, the melting point of which was not less than 147°C.

4) Concentrated sulphuric acid.

Procedure: To the citrate solution containing the dissolved beta-cholesterol (vide supra) 0.3 ml glacial acetic acid, 1.5 ml/p-toluenesulphonic acid and 4.0 ml of acetic anhydride were added without mixing. The solution was allowed to stand at room temperature until it had cooled and then 0.4 ml of concentrated sulphuric acid was added, after which the solution was thoroughly mixed until the precipitate had completely dissolved. The optical density was measured using the Evelyn photometer with the 565 mμ filter and the 6mm aperture, the solution having stood at room temperature for 20 minutes.

A standard equivalent to 100% beta-cholesterol was set up by replacing the sodium citrate with water without adding glacial acetic

acid and was treated as above. A blank was also made replacing the sodium citrate with water and the same procedure was followed.

The method of Pearson, Stern and McGavack was frequently used to check the results of determinations of the serum total cholesterol by the technique of Sperry and Webb.

The alpha-cholesterol levels were estimated by subtraction of the beta-cholesterol from the total cholesterol.

iii. Phospholipids

The amount of serum phospholipids was determined by analysis of the lipid phosphorus, and using the coefficient 25.0 the phosphorus value was converted to phospholipids. After burning an ethanol-ether extract, lipid phosphorus was determined by the method of Fiske and Subbarow (1925).

Reagents:

1) Sulphuric acid, 10N: 450 ml concentrated sulphuric acid were added to 1300 ml of water.

2) Molybdate I - 2.5% ammonium molybdate in 5N sulphuric acid. In 200 ml distilled water 25 gm of the salt were dissolved. It was rinsed into a litre volumetric flask containing 500 ml of 10N sulphuric acid. It was then diluted to the litre mark with water and mixed.

3) Molybdate III - 2.5% ammonium molybdate in water. As soon as any considerable amount of sediment (ammonium trimolybdate) appeared, the solution was discarded.

4) Standard Phosphate: the solution which was made up

contained 0.4 mg phosphorus in 5 ml of the solution. 0.3509 gm of pure monopotassium phosphate were dissolved in water. The solution was then transferred quantitatively to a litre volumetric flask, 10 ml of 10N sulphuric acid were added and it was finally diluted to the 1 litre mark with water and was mixed.

For the working standard 10 ml of the above stock were taken and diluted with 100 ml distilled water.

5) 15% Sodium Bisulphite: 150 gm sodium bisulphite was dissolved in 100 ml of water. Care was taken to see that the solution was free of turbidity before it was utilised.

6) 20% Sodium Sulphite: 20 gm of the crystalline sulphite were dissolved in 380 ml of water. Any suspended matter was removed by filtration.

7) Reducing Agent: 0.9 gm of 1-amino-2-napthol-4-sulphuric was added to 390 ml sodium bisulphite solution. It was dissolved by heating and allowed to cool. Another 10 ml sodium sulphite solution was added. The solution was filtered seven times with a Wattman No.1 filter. It was stored in a cupboard, or in a dark bottle.

8) Other reagents used were hydrogen peroxide, 5N sulphuric acid and Bloor's solution (1 part ethyl ether to 3 parts of ethyl alcohol).

Procedure: One ml of serum was added to a 25 ml

volumetric flask half filled with Bloor's reagent. Having rinsed the sides of the flask the contents were boiled, cooled and brought up to volume at 25 ml and then filtered through a Wattman No.1 filter. Two aliquots of 10 ml were taken and pipetted in Kheldahl flasks. They were evaporated to dryness and 2.5 ml 5N sulphuric acid was added. The extracts were placed in an oven at 160°C for 2 hours until they were black, eight to ten drops of hydrogen peroxide were then added, after which they were returned to the oven for 20 to 30 minutes. After cooling the extracts were then transferred into wide necked 25 ml volumetric flasks with 18 ml water and 2.5 ml of molybdate III and 1 ml of reducing agent were finally added.

Standards and blanks: 2 ml, 4 ml, 6 ml and 8 ml of working standard solution were placed in four 25 ml volumetric flasks. 2.5 ml of molybdate I was added to the blanks and standards.

To all flasks, i.e. blanks, standards and serum extracts, 1 ml of reducing agent was added. The solutions were then made up to volume with water. After 15 minutes all samples were read off an Evelyn colourimeter at 660 mμ with a 6mm aperture.

All samples collected from a single subject over the year were analysed at one session for their lipid content, thus eliminating chance errors due to technique which might have occurred should the block of samples for a particular month have been estimated at one sitting. The probability of missing a seasonal or monthly effect was thereby greatly diminished.

Figure 8. The fasting serum total lipid concentration for all subjects taking part in the Survey. The central continuous line represents the monthly mean, and the outer interrupted lines demonstrate the standard deviations.

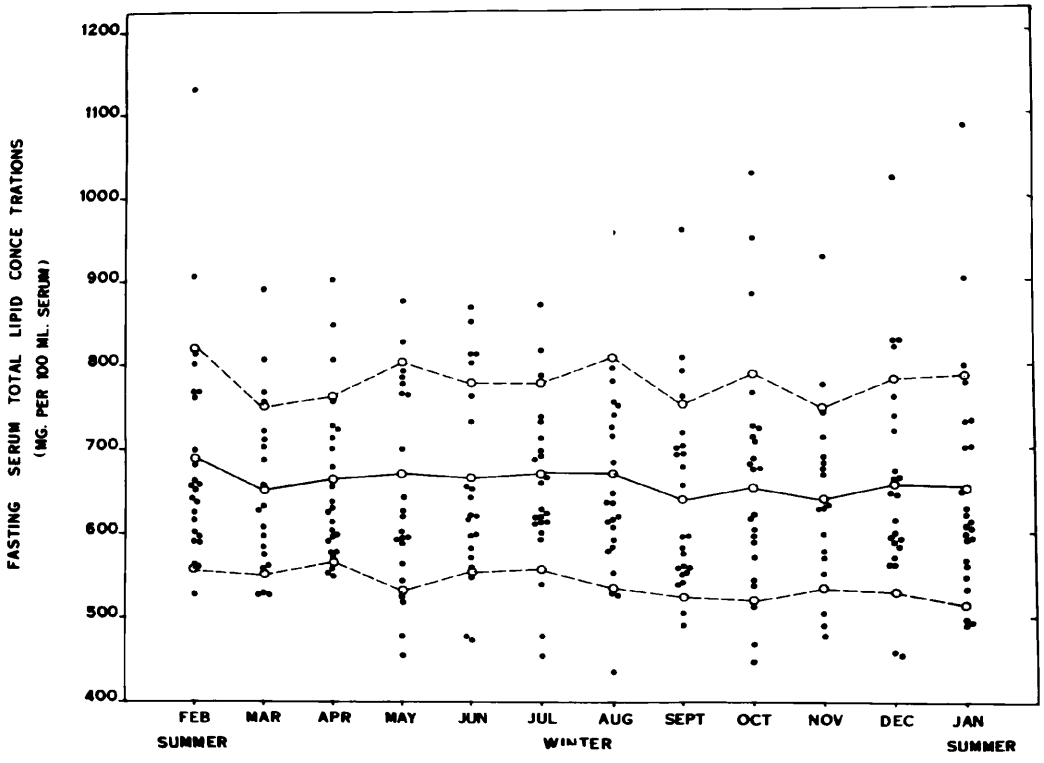


Table 18. The mean monthly changes in serum total lipids, with standard deviations (mg/100 ml serum)

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Mean	687.9	651.5	659.7	665.9	664.8	666.0	668.6	639.4	652.6	640.1	655.6	650.2
S.D	130.0	99.5	98.8	136.0	113.0	111.0	138.0	112.0	135.0	107.0	126.0	135.4
Grand Mean	655.6 ± 122.0											

B. VARIATIONS BETWEEN SEASONS

1. Total Lipids and Total Fatty Acids

a) Results

The grand mean and standard deviation for all levels of serum total lipids during the year was 655.6 ± 122.0 mg %. The results of the individual values at monthly intervals have been demonstrated in the form of a scattergram in Figure 8, the central continuous line representing the monthly mean and the interrupted outer lines indicating the mean monthly levels ± one standard deviation. The numerical values for the monthly means and standard deviations have been shown in Table 18. The maximum mean level of 687.9 ± 130 mg % occurred in the first month, which was followed by a drop of 36 mg to a level of 651.5 ± 99.5 mg % in the second. During the months April to August there was a slight elevation on the March level, and the last 5 months showed lower readings than those in the first 7 months. (From February to August, the mean was 666.3 and from September to January the mean was 647.6 mg %). The levels for the months April to August which approximate to the period of continuous darkness and lowered temperatures were slightly raised when compared with the March reading.

Table 19. Shows the mean levels of the serum total lipid for the months February and April for the first and second year subjects and for the complete group of subjects.

	February	April
First year subjects	694.7	650.4
Second year subjects	686.9	678.3
Whole group	687.9	659.7

From Table 19 it can be concluded that the group of first year men

Table 20. The mean monthly changes in serum total fatty acids, with standard deviations (mg/100 ml serum)

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Mean	398.2	315.5	376.9	389.1	387.8	373.4	391.0	358.5	379.7	369.7	386.8	387.9
S.D.	84.4	62.9	68.1	103.0	78.4	89.4	102.0	79.8	103.0	79.8	105.0	108.0
Grand Mean	383.2 ± 88.3											

demonstrated the greater fall in total lipid levels in comparison with those who had already passed a year in Antarctica, between the months February and April.

The monthly means and standard deviations for the serum total fatty acids are shown in Table 20. The grand mean amounted to 383.2 $\pm$ 88.3 mg %. There was a marked fall in levels during March and April, similar to the changes in the serum total lipid levels during these months, but, apart from this finding, there was no monthly trend suggestive of a seasonal effect in the data.

b) Comments

The mean total lipid concentration was lower than the level reported by Schrade, Boehle and Biegler (1960) in twentythree young males, namely 708 mg %. The causation of the highish result in February for total lipids and for total fatty acids is difficult to establish. Whether this represented a previous value for the group, and the increased activity which occurred at this time of the year caused the drop, or whether it was dependent upon the high calorie intake, cannot be definitely stated. It is probable that an increase in body weight was occurring during February, though there is no direct evidence for this as the first estimations of body weights were made on the same days as the first blood sampling. However, the mean weight showed an increase over March and April and the total lipids were substantially depressed during these months. Eight of the subjects had already spent a year at the base and to compare the means of the first year group with that of the second year group helps to clarify this situation. The large fall which occurred in total lipid levels in the first year subjects, coupled with the fact that at this time they were in positive calorie balance was highly suggestive that the cause

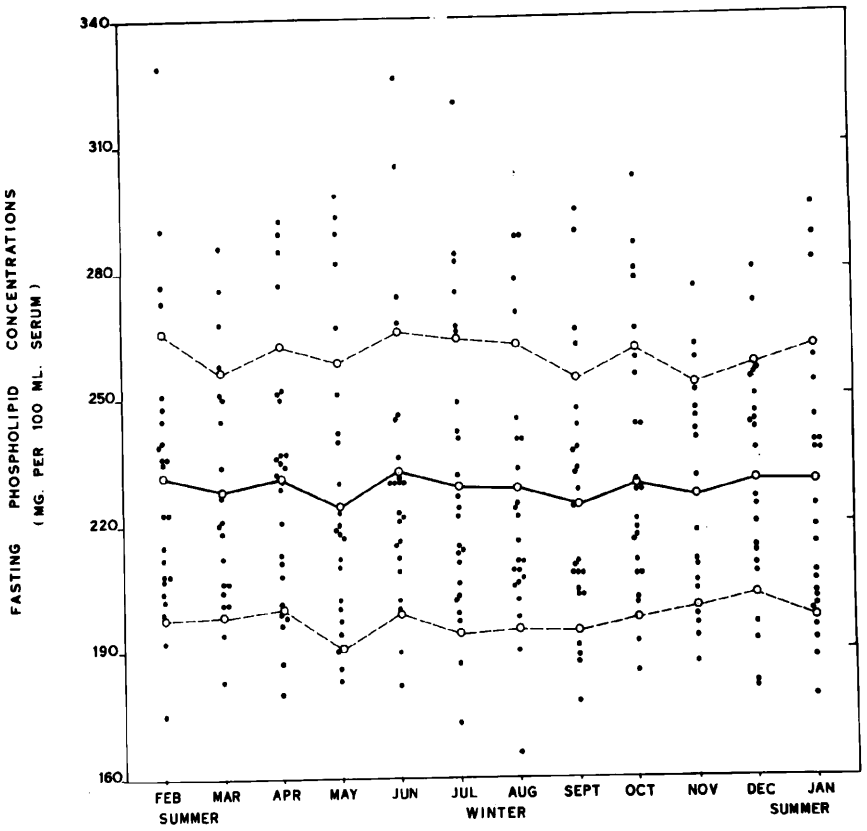


Figure 9. The monthly changes in serum phospholipid concentrations for 24 subjects. The central continuous line shows the monthly means, and the outer interrupted lines represent $\pm$ 1 S.D.

Table 21. The mean monthly changes in serum phospholipid levels, with standard deviations (mg/100 ml serum)

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Mean	231.9	227.8	231.3	224.5	232.3	229.0	228.6	224.5	229.5	226.4	230.1	229.5
S.D.	34.0	28.6	31.3	34.0	33.3	34.9	33.7	29.9	31.9	26.3	27.2	32.0
Grand Mean 221.4 ± 20.7												

could be attributed to either the increased physical activity, or to an increase in the exposure to cold.

2. Serum phopholipids, free and total cholesterol, with cholesterol esters and the cholesterol:phospholipid ratio

a) Results

The monthly means and standard deviations of the phospholipids are shown as a scattergram in Figure 9. The grand mean and standard deviation was 221.4 ± 20.7 mg %. No monthly trends were discerned over the year (see Table 21). There was no apparent fall between the months February and April, and no elevation of the means for the first 6 months, over the means of the second 6 months.

The grand mean for total cholesterol levels in the year was 199.0 ± 31.7 mg %. The monthly means and standard deviations are demonstrated in Table 22 and shown in graph form in Figure 10. There was a fall of 9.8 mg from the initial February mean of 209.8 to the April mean of 200.0 mg %. The results for these 2 months were treated in a similar way to that employed for the same months for the total lipid levels, and changes in mean levels for the first and second year men were compared. The group differences have been tabulated in Table 22. A fall of 12.8 mg occurred between February and April in the serum cholesterol of the first year men, and one of 2.8 mg % was apparent in the serum cholesterol of the second year subjects. From Table 22 it can also be seen that there was a far greater overall depression over the 12 months of the survey in the first year subjects than in the second year subjects; the decrements amounted to 24.2 and 8.3 mg cholesterol/100 mg serum respectively (compare Figures 15 and 16).

Figure 10 shows that there was an overall tendency for the

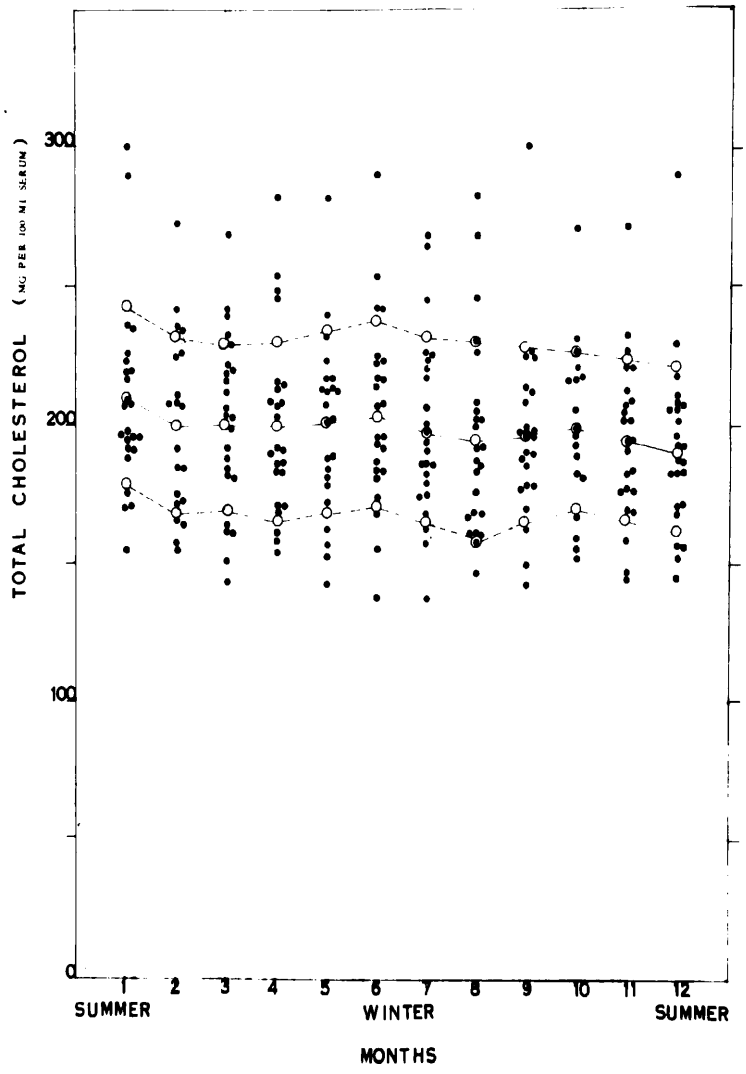


Figure 10. The scattergram of the individual total cholesterol levels at monthly intervals over the period of one year. The central continuous line shows the monthly means, and outer interrupted line demonstrates $\pm$ 1 S.D.

Table 22. The monthly means of serum total cholesterol levels for the group, and for first and second year subjects.

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Monthly mean for whole group	209.8	199.8	200.0	199.8	200.7	204.4	198.7	194.5	196.3	198.4	194.3	190.9
S.D.	33.0	28.6	31.4	31.9	37.5	32.5	31.7	34.3	31.6	29.7	28.4	29.6
Means for 1st yr subjects	211.3	197.1	198.5	200.1	201.9	205.6	197.6	193.3	195.8	199.3	191.9	187.1
S.D.	42.0	36.3	34.1	39.9	39.9	36.7	36.6	41.7	36.7	35.9	32.9	34.9
Means for 2nd yr subjects	206.9	206.3	204.1	199.2	198.5	201.9	200.8	197.0	197.4	201.5	199.0	198.6
S.D.	36.5	26.7	16.9	16.3	22.2	18.1	24.6	24.3	21.1	24.3	17.1	12.5
Grand mean	199.0	± 31.7										

mean cholesterol levels for the whole group to fall during the year. A statistical analysis was made of the data to find the significance of the monthly trends suggested from the meaned results, with a view to relating these trends to dietary intake, physical activity and changes in body weight. Twelve monthly readings should have been available for each of the twentyfour subjects. Unfortunately a number of observations were missing due to periodic absence of men who were taking part in sledging journeys. In order to simplify the analysis the readings corresponding to the months March and November were omitted completely, and further, only twenty subjects were considered, subjects SM, MJ, DED and MT being eliminated from the analysis. The remaining data formed a complete block, a single observation relating to a single subject each month. Initially each response was treated separately. A two way analysis of variance was carried out on the data.

The analysis of variance indicated that the month effects differed significantly at the 0.1% level amongst themselves and therefore from zero. The estimated overall month and subject effects have been shown in Tables 97 and 98 in the Appendix to Section VI, together with an estimation of the appropriate variance which is a measure of the precision of the estimates.

The estimates of the month effects suggested that during the period August to January, the total cholesterol levels were substantially reduced. The average effect for the months February to July less the average effect for the months August to January was compared with zero by means of the Scheffe comparison technique. It was found to be significant at the 5% level.

The grand mean and standard deviation for the cholesterol: phospholipid ratio was 0.87 ± 0.09. The scattergram of the individual

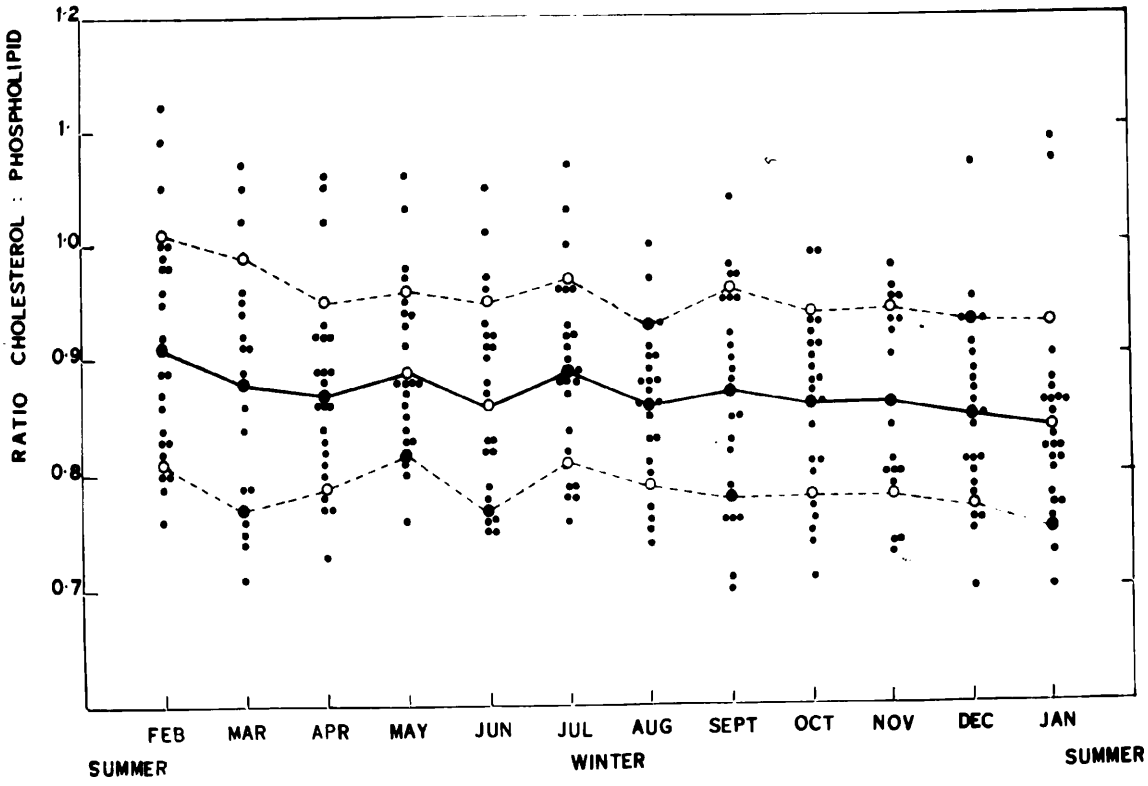


Figure 11. The individual values for the cholesterol: phospholipid ratio at monthly intervals, with monthly means and standard deviations.

Table 23. The mean monthly changes in the C:P ratios for the entire group, with standard deviations, and the mean monthly changes for first and second year subjects. The grand mean for all results is also shown.

	Feb 1961	Mar	April	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan 1962
Monthly means for group	0.91	0.88	0.87	0.89	0.86	0.89	0.86	0.87	0.86	0.86	0.85	0.84
	±0.10	±0.11	±0.08	±0.07	±0.09	±0.08	±0.07	±0.09	±0.08	±0.08	±0.08	±0.09
Monthly means for 1st yr subjects	0.91	0.88	0.86	0.89	0.86	0.90	0.86	0.87	0.86	0.86	0.86	0.85
Monthly means for 2nd yr subjects	0.89	0.87	0.88	0.88	0.86	0.88	0.86	0.85	0.85	0.85	0.83	0.82
Grand Mean	0.87	± 0.09										

readings, with the monthly means and standard deviations are shown in Figure 11 and in Table 23. There was a distinct tendency for the cholesterol:phospholipid ratio to fall during the year. This lowering was largely due to the depression of the total cholesterol levels, which was not simulated in the phospholipid values. It has been accepted previously that changes in serum cholesterol are usually followed by quantitatively equal alterations in serum phospholipid and it is interesting that in the present survey there was evidence that this is not entirely the case. The initial readings of the C:P ratio in February was 0.91 and the final value in January was 0.84.

On examination of the C:P ratios for the two groups, the newcomers and the second year subjects, it was found that they both demonstrated a downwards trend during the year which were of the same order of magnitude.

The grand mean and the standard deviation of free cholesterol was 49.1 $\pm$ 10.2 mg%. The monthly means with standard deviations for all results have been demonstrated in Table 24. Apart from the rise of 4.4 mg % in the levels between February and March, the levels showed little fluctuation and no overall trend or any suggestion of a seasonal change was indicated.

The grand mean and the standard deviation of the cholesterol esters was 150.2 $\pm$ 24.6 mg %. It should be noted that 75.4% of the total cholesterol was in the form of the ester. There was a well marked fall of 14.2 mg % between the first and second months of the survey and there was a tendency for levels to fall over the year (Table 25). It is clear that the cholesterol esters showed the same changes as were demonstrated by the total cholesterol levels.

Table 24. The mean monthly changes in serum concentrations of free cholesterol (mg/100 ml serum)

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Mean	44.7	49.1	48.8	45.1	49.4	49.8	51.1	51.7	51.0	52.6	49.5	47.7
Standard Deviation	±9.7	±9.2	±8.7	±11.1	±10.7	±10.6	±10.4	±10.7	±9.5	±10.7	±9.5	±10.0
Grand Mean	49.1 ± 10.2											

Table 25 . The mean monthly values of the serum cholesterol ester content in mg/100 ml serum, with standard deviations

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Mean	165.2	150.8	151.9	154.7	151.3	154.5	147.7	143.9	145.1	147.4	145.6	143.2
Standard Deviation	±26.0	±24.2	±23.7	±24.9	±24.8	±25.2	±24.0	±26.9	±25.0	±21.7	±23.3	±21.8
Grand Mean	150.2 ± 24.6											

b) Comments

The grand mean of the phospholipid results agrees with that reported by Havel, Elder and Bragdon (1955) who carried out a survey of young males, namely 218 mg %, and also with the average level reported by Foldes and Murphy (1946) of 227 mg %. The cholesterol:phospholipid ratio was on the high side and this largely was due to the slightly raised total cholesterol level. Havel et al reported a C:P ratio of 0.79 for male subjects within the same age group as the subjects of the present survey.

The grand mean of all total cholesterol levels is slightly above that reported by Keys, Mickelson, Miller, Hayes and Todd (1950) for one thousand and fortyseven normal American males of average age 22.1 years, ranging from 17 to 30 years, the mean total cholesterol value of whom was 178.8 $\pm$ 33.9 mg %. It agrees more closely with the data of Barker (1939) who ascertained that the mean total cholesterol level of American males in the third decade was 203 mg % and with that quoted by Bronte Stewart (1959), namely a mean level of 197.7 mg %. Peters and Man (1943) reported a mean total cholesterol level of 194.1 mg %. Tanner in 1951, measured cholesterol levels in Englishmen of 18 to 36 years and found it to be an average of 190 mg % and Keys, Fidanza, Scardi, Bergami and Keys (1954) found the cholesterol concentration in young Neapolitan males to be 210 mg %. The mean total cholesterol was reported to be 189 mg, S.D. = 34 mg %, in 72 Illinois medical students (Peeler, Hepler, Kinney, Cisler and Jung (1950-51) and was 186 $\pm$ 26 in ten men and ten women between 19 and 35 years in Boston (Foldes and Murphy, 1946). Consolazio and Forbes (1946) found that the mean for twelve men in the same age range in Boston was 182 mg %. All of these subjects seemed to be in good health and presumably subsisted, on the

average, on the relatively high fat diet customary in the United States and in Great Britain. For thirty Danes of from 19 to 30 years of age Kornerup (1950) reported an average of 195 mg %; these subjects presumably subsisted on the common diet of Denmark which also is relatively high in fat. In all groups of healthy young men studied by comparable methods, therefore, a mean value of about 200 mg cholesterol % derived from casual blood sampling was the common result. Karvonen, Rautanen, Rikkonen and Kihlberg (1958) estimated the serum cholesterol levels in fortyfour male Finnish champion skiers and they reported that the mean level was 204 mg %. The mean age of the group was 28.1 years. Karvonen pointed out that the mean value was lower than that of the surrounding population and suggested that it was the severe exercise which the group frequently experienced which caused them to have this lower level. The mean total cholesterol value for the Halley Bay subjects was close to that of the surveys reviewed.

It is difficult to account for the slight downward trend in monthly mean levels of the serum total cholesterol levels, which was demonstrated in a more exaggerated form by the monthly mean levels of cholesterol ester. The fall shown by the first year subjects was greater than that of the second year subjects. The implication is that the previous "ship" level was influenced either by climatic factors or by the more direct factors of energy intake and energy expenditure. The cholesterol values showed no tendency to follow the seasonal changes in the total calorie or fat intake. The mean body weight showed the greatest rise during the first 2 months for both first year and second year subjects, yet for the first year subjects the March total cholesterol value was the lowest for the first 6 months of the year. It appears improbable that serum cholesterol levels reflected a positive or negative

calorie balance under the circumstances of the present experiment, and it appears that they were either related to physical activity per se, or to the outside exposure with the possible addition of cold stress.

A slight tendency was shown for winter levels to rise in monthly values for all subjects and in the means for the first year and second year men. The survey by Keys et al(1958) on Finlanders showed a winter rise amounting to 100 mg % in total cholesterol levels but no significance was attached to this marked rise beyond suggesting that it was due to environmental factors. In a diet survey reported by Ronic, Pekkarinen, Karvonen and Kihlberg (1958), using the same subjects as those used for the Finland serum lipid survey, there was a minimal difference between intakes for winter and summer. It may be that during the winter the East Finlanders reduced their activity in the same way as the Halley Bay subjects, but without lowering their food intake. It is interesting that subjects in the Antarctic showed only a suggestion of a winter rise, particularly when it is seen that the seasonal changes, in the locality of Finland where the survey was carried out, were similar to the seasonal changes at Halley Bay. Paloheimo (1961) also noted a seasonal trend in serial cholesterol concentrations in a group of convicts in Finland, but could not account for the changes. He reported similar changes in serum phospholipid levels. In a group of policemen, Paloheimo could discern no seasonal changes in serum lipid levels. Reference should be made to the latter pages of the introduction for the results of other workers concerning seasonal changes in total cholesterol concentrations.

Much has been said and written about psychological stress during the winter darkness in isolated communities in polar regions, and a psychological element cannot be dismissed as a causative factor for the

very slight winter elevation. Friedman, Rosenman and Carrol (1958) measured the effect of socioeconomic stress upon serum cholesterol levels in accountants and concluded that such stress was marked by sudden and often profound rise in cholesterol concentrations. The difference between levels at maximum and minimum stress was, in this expriment, 42 mg % in serum cholesterol levels, which was far greater in amplitude than the winter rise shown in the subjects at Halley Bay.

It is possible that the downwards trend, more marked in the first year subjects, might have represented a slow process of acclimatization to cold. The premise that the second year men were already acclimatized to cold is supported by the minimal fall shown in their cholesterol levels. To infer that serial blood cholesterol levels provide an index of thyroid function is an over simplification but the changes in the levels are difficult to explain wholly on the basis of food and fat intake, or of energy expenditure.

The connection between the thyroid gland and serum cholesterol levels was first noticed by Epstein and Lande in 1922. They noted that the total serum cholesterol was decreased in patients with hyperthyroidism and increased in patients with hypothyroidism. Histologically, the thyroid shows increased activity in animals exposed to cold (Starr and Roskelley, 1940). Le Blond and Gross (1943) found evidence of increased thyroid function with radioactive iodine studies in groups of cold exposed rats. The thyroid fixed and metabolised iodine greatly in excess of controls during a period of 7 to 26 days after the initiation of the experiment. After 40 days, iodine metabolism was back to control levels and the histological picture was similar to the controls. A seasonal difference in response to the administration of thyroxine in animals had been shown by Mansfeld and Scheff-Pfeiffer (1938). The effect of thyroxine diminishes in the

warm season and increases in the cold, and this effect is said to be due to the secretion in excessive heat of thermothyrine which inhibits the metabolic effect of thyroxine (Mansfeld et al, 1938; Mansfeld, 1942). Thus there is evidence of hormonal acclimatization to cold in animals but it must be admitted that data reported as evidence for acclimatization to cold in man is somewhat scant. It seems paradoxical that in the winter months serum cholesterol should show a slight rise indicating a reduced thyroid output but in effect the environment is changed to that of a temperate one as a result of the enforced and continued lack of outside exposure.

In the case of the Halley Bay subjects, it can only be said that the decrease shown in the newcomers, which was greater than that of the second year subjects, may have represented the cumulative effect of low environmental temperature acting upon the endocrine system and stimulating the thyroid gland to cause an increase of metabolic rate. However, the pulse rates measured at monthly intervals showed little indication that they increase with the length of stay in Antarctica, and therefore the view that the trends of the cholesterol levels indicated that there was an increased secretion of the thyroid gland falls down on further critical assessment.

Oliver and Boyd (1957) uphold that the approximate range of cholesterol ester in normal individuals is 80 to 160 mg % and that free cholesterol lies between 40 and 90 mg %. The mean level of free cholesterol appeared to be on the low side in the Halley Bay survey and the cholesterol ester level to be slightly raised. The percentage of the total cholesterol in the form of the ester was 75%, whereas, using the median points of the ranges reported by Oliver and Boyd (1957), the percentage of esterified cholesterol would have been 65%. However,

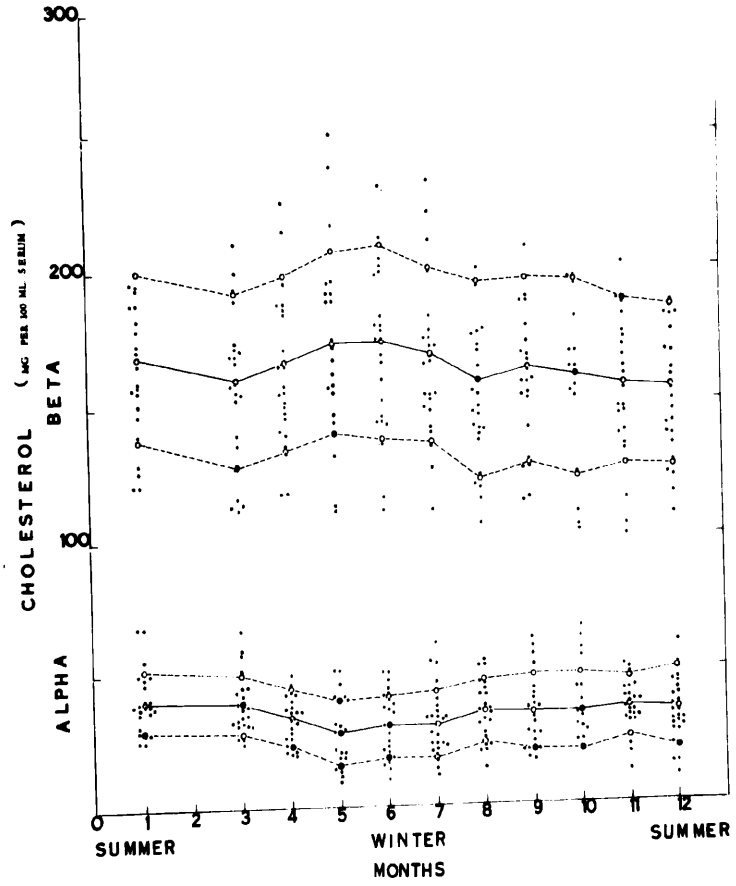


Figure 12. The individual monthly values of alpha- and beta-cholesterol concentrations for 24 subjects at Halley Bay. The upper portion demonstrates the beta-cholesterol levels, and the lower portion shows the alpha-cholesterol levels. Demonstrated with monthly mean values and with standard deviations.

Table 26. The monthly changes in serum β-cholesterol levels for the group, and for the first and second year subjects.

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Means for the entire group	169.0	-	160.0	166.0	173.9	174.4	168.9	159.5	163.0	161.3	157.5	155.5
S.D.	31.6	-	32.7	32.2	33.7	36.1	32.3	36.4	34.6	36.5	30.3	29.7
Means for 1st yr group subjects	167.4	-	157.8	166.6	173.9	176.1	170.6	159.9	161.5	159.8	152.5	148.4
S.D.	17.1	-	37.0	36.3	42.7	44.4	37.9	42.3	39.5	42.7	32.1	35.6
Means for 2nd yr group subjects	172.3	-	164.9	165.0	172.2	170.8	164.3	158.8	167.6	164.8	167.5	169.5
S.D.	19.4	-	15.2	19.7	22.6	17.7	20.0	23.0	7.9	36.3	14.8	6.8
Grand Mean	165.0 ± 33.6											

it is closer to the result of Foldes and Murphy (1946) of 69% cholesterol ester. This suggestion of an elevated cholesterol ester level is difficult to explain; it is possible that during the storage of the original samples the serum free fatty acids may have been taken up by the cholesterol groups.

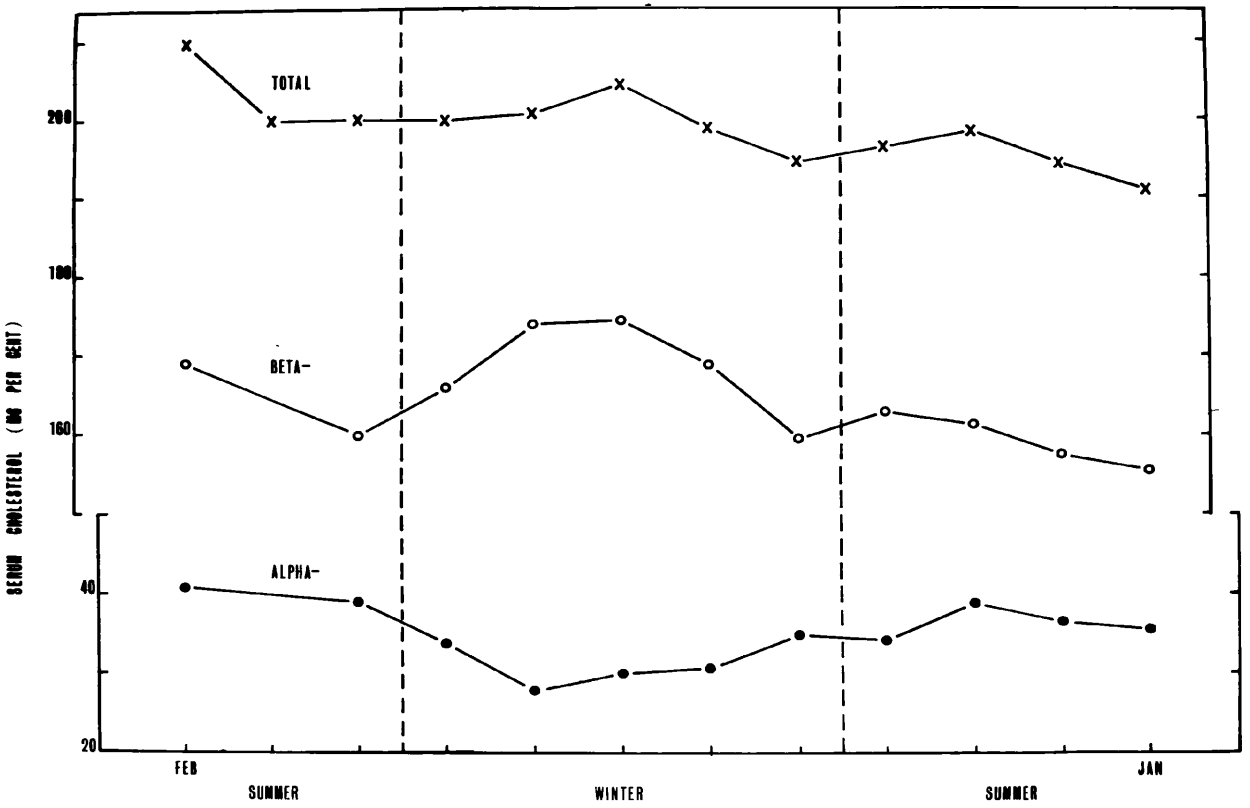
3. Alpha- and Beta-cholesterol

a) Results

The grand mean and standard deviation of all serum beta-cholesterol levels was 165.0 $\pm$ 33.6 mg %. The monthly means and standard deviations are shown in Figure 12 as a scattergram and in Table 26 as meaned monthly values. Figure 12 indicates that there was a fall in beta-cholesterol levels of 9 mg % between February and April. If these figures are examined for the effect of the two groups, namely first and second year subjects (Table 26 and Figures 15 and 16), it can be seen that a greater overall depression of levels occurred in the first year subjects than in the second year subjects thus showing that there were similar group responses as in the case of the serum total cholesterol estimations.

From Figures 12 and 13 it is seen that there was a tendency for the beta-cholesterol mean to rise during the months May to July, and then to fall during the months August to September. The month effects were tested by the analysis of variance, using the same subjects employed for the statistical analysis of the total cholesterol levels with the omission of the same months. The month and men effects were found to differ significantly at the 0.1% level. Tables 97 and 98 in the Appendix show the estimated effects. A phenomenon similar to that found in the total cholesterol analysis was suggested. Consideration of a contrast involving the differences between the mean effect

Figure 13. The mean changes in total cholesterol, and alpha- and beta-cholesterol at monthly intervals over the year, for all subjects.



for the first 6 months and the mean effect for the last 4 months, using the Scheffe comparison technique, substantiated the difference at the 5% level. The winter rise which occurred in the mean values cannot be said to have been a significant change. That the winter elevation suggested from the results was not significant must be dependent on the fact that the February evaluation for the beta-cholesterol was somewhat high, corresponding to the elevated total cholesterol reading for the month.

The grand mean and standard deviation for beta-cholesterol when expressed as a percentage of the total was 81.8 $\pm$ 7.3 mg %. The monthly means and standard deviations are shown in Table 27 and as a scattergram in Figure 14. It can now be seen that when the beta-cholesterol levels were calculated as a percentage of the total cholesterol, the downwards trend of the total cholesterol which was reflected in the absolute beta-cholesterol levels was eliminated and there was an elevation shown in the winter months. It is clear that during the months May to October, the levels were elevated over those for the remaining months.

The data was tested by two-way analysis of variance for the significance of the month and the man effects. The same group of subjects was employed for beta-cholesterol % analysis as was utilised for the serum total cholesterol levels. It was found that the month effect was significantly different from zero at the 0.1% level. The comparison of values for the months of June, July and August, less those for the months February and April, and December and January was significant at the 0.1% level.

The alpha-cholesterol grand mean and standard deviation was 34.7 $\pm$ 12.9 mg %. Referring to Figure 12 it can be seen that there was

Figure 14. The beta-cholesterol expressed as a percentage of the total cholesterol, for each of 24 individuals at monthly intervals. The central continuous line represents the monthly mean, and the outer interrupted lines demonstrate the standard deviation.

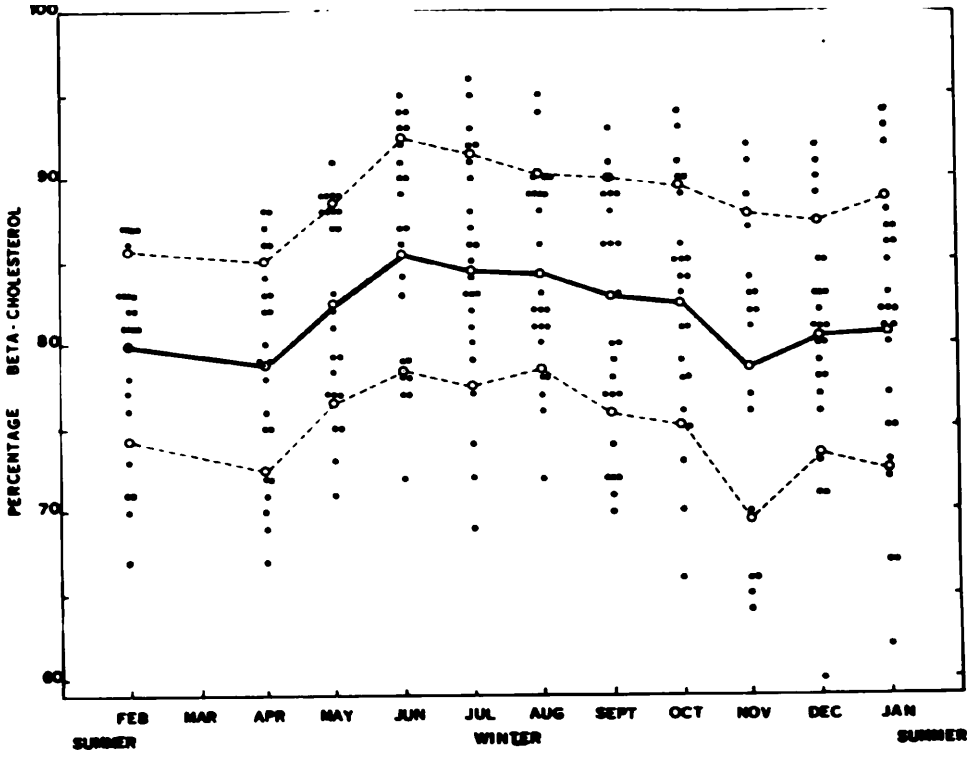


Table 27. The mean monthly changes in serum β-cholesterol levels, expressed as a percentage of the total cholesterol.

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Mean	79.9	-	78.8	82.5	85.4	84.5	84.3	82.9	82.4	78.7	80.4	80.7
S.D.	5.7	-	6.3	6.0	7.0	7.0	5.9	7.1	7.2	9.1	7.0	8.2
Grand Mean	81.8 $\pm$ 7.3											

Table 28. The mean monthly changes in serum α-cholesterol levels, with standard deviations (mg/100 ml serum)

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Mean	40.8	-	39.0	33.8	27.9	30.0	30.7	35.0	34.2	38.9	36.5	35.5
S.D.	11.4	-	10.8	10.6	12.8	11.7	12.6	11.4	13.7	14.2	11.1	15.1
Mean levels for 1st yr subjects	43.8	-	39.6	33.6	28.7	29.5	27.8	33.4	34.7	40.7	39.7	38.6
S.D.	12.0	-	9.9	12.6	10.9	13.4	10.6	12.9	12.8	14.1	10.7	15.8
Mean levels for 2nd yr subjects	34.9	-	39.3	34.3	26.3	31.0	36.4	38.3	29.9	34.6	31.5	29.1
S.D.	4.8	-	9.2	12.2	14.8	12.3	16.1	12.8	15.7	12.7	6.9	11.1
Grand Mean	34.7	$\pm$	12.9									

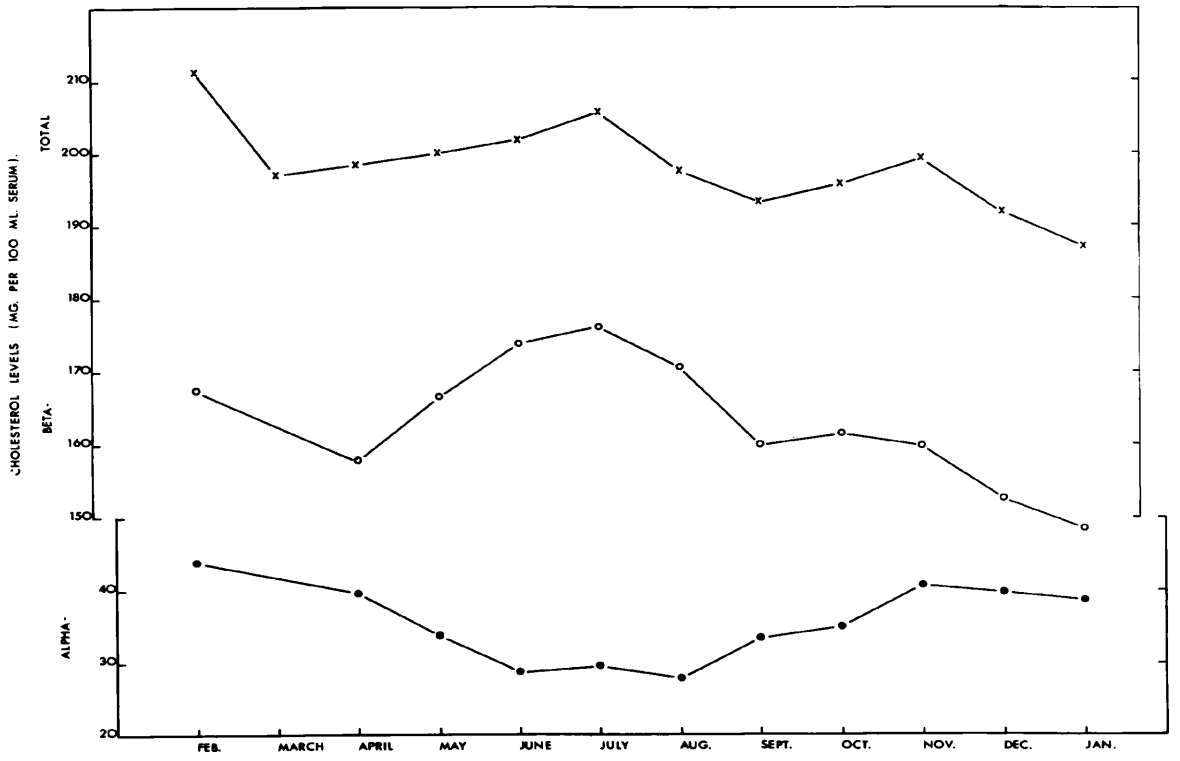
a tendency for the alpha-cholesterol levels to be depressed during the months May to September. In Table 28 the monthly means and standard deviations have been shown. The readings for the last 4 months of the year were slightly lower than those for the months February and April. Thus, the trend of the total cholesterol was to all appearances simulated in the alpha-cholesterol levels.

The alpha-cholesterol levels were examined using variance analysis in a search for a seasonal and a man effect. Both were found to be significant at the 1% level. The estimates of the month effects (Table 97 and 98) suggested that there was a depression during the months, June, July and August. An appropriate contrast was considered and was found to be significant at the 5% level. Similarly, the slightly reduced values at the end of the year were compared with those values of the alpha-cholesterol at the beginning of the year, and were found to be non-significant. The slight depression shown during the winter months was therefore significant for absolute alpha-cholesterol levels. It is conceivable that if the downwards trend of the total cholesterol had not occurred, then the beta-cholesterol would also not have followed this trend, the winter elevations would have occurred from a 'horizontal' level and would probably have been significant.

From Figures 13, 15 and 16 it becomes clear that the larger group of first year subjects influenced the changes in serum levels of the whole group to a greater extent than the second year subjects. The levels of the second year subjects tended to mask the changes in the levels of the group, even though a certain degree of significance was established for the changes in levels for all subjects.

In the second year group, the winter rise in the beta-cholesterol with the depression in alpha-cholesterol was not distinct (see Figure 16). No reason can be given for this group difference.

Figure 15. The mean monthly changes in total cholesterol (x), beta-cholesterol (o), and beta-cholesterol (.) levels during one year at Halley Bay for first year subjects.



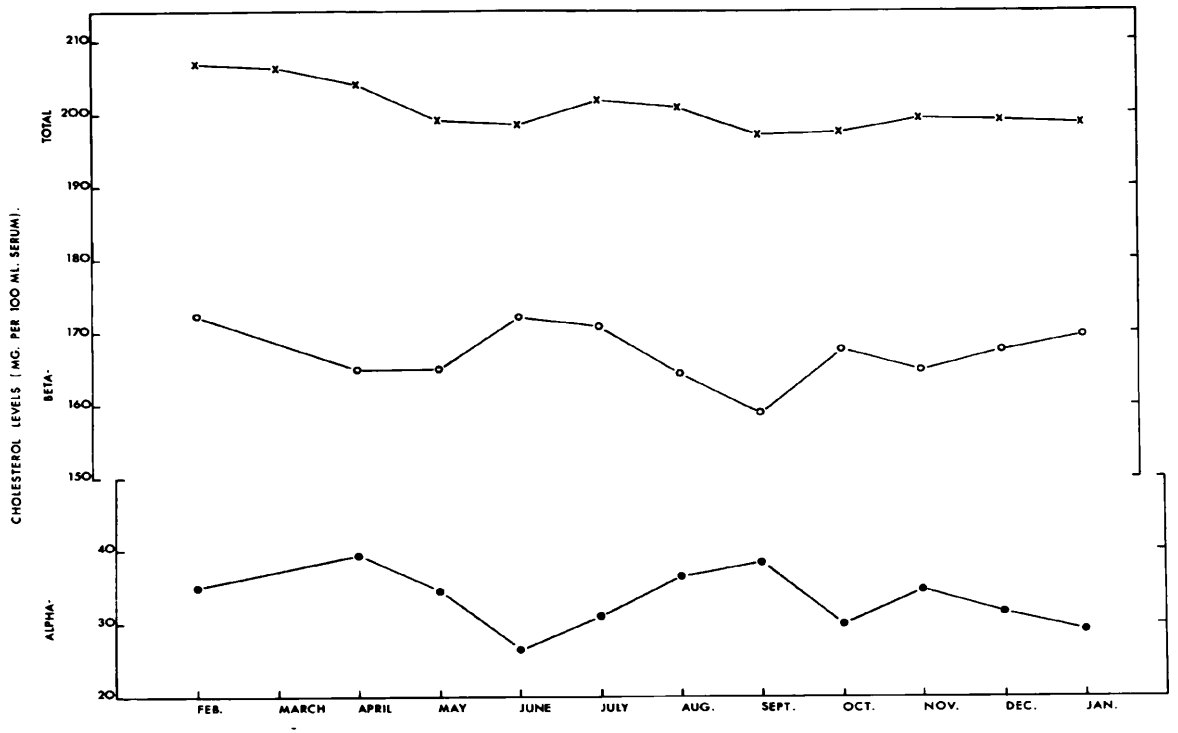
It seems plain, however, that the depression in total cholesterol and beta-cholesterol concentrations between the first and last month of the survey for the first year subjects was greater in magnitude than that for the second year subjects. It has been mentioned that a large proportion of this fall occurred between the months February and April.

b) Comments

The mean alpha-cholesterol level of 34.7 mg% was on the low side when compared with the results of many workers. Oliver and Boyd (1957) reported that the approximate range of alpha-cholesterol in health lay between 40 and 80 mg % and Keys et al (1958) pointed out that the mean level in a group of males in social classes 3 and 4 was 44.0 mg %. The Halley Bay result was considerably lower than that of Waris (1958) which ranged between 55 and 57 mg % and that of Konttinen (1959) at 56 mg %. The ratio of alpha-cholesterol to total cholesterol at 18.2 % was low when compared with that reported by Konttinen (1959) at a level of 27%. It follows that the proportion of cholesterol in the beta-lipoprotein was elevated at 81.8%. Bronte-Stewart (1959) reported that 75% of the total cholesterol was attached to the beta-lipoprotein in the age group 18 to 35 years.

The alpha-cholesterol levels were higher during the summer and lower in the winter months. The beta-cholesterol showed the opposite tendency and the statistical significance of the variation of the beta-levels was found to be negative for the seasonal effect because of the overall downwards slope shown in these levels. The summer months were associated with greater physical activity and increased food intake when compared with the winter months (Table 32). Thus it follows that the fluctuations in alpha- and beta-lipoprotein cholesterol values were the result of either variations in food intake,

Figure 16. The monthly changes in total cholesterol (x), beta-cholesterol (o), and alpha-cholesterol (.) levels for the second year subjects at Halley Bay.



or changes in energy expenditure and 'outside exposure'.

The seasonal changes shown in the lipoproteins at Halley Bay were similar to those found by Paloheimo (1961) on a group of convicts in the Northern Hemisphere. He reported that total cholesterol levels were maximum in January and minimum in May, beta-cholesterol was maximum in January and minimum in April, and alpha-cholesterol/total cholesterol ratio was highest in April and lowest in December.

Antonis and Bersohn (1962) in their experiments on Bantu and European prisoners found that on increasing the percentage of calories supplied by fat from 15 to 40%, the total cholesterol was markedly elevated, and that this rise occurred in the beta-cholesterol fraction while the alpha-fraction remained unchanged. In the Halley Bay subjects, the winter rise in the beta-cholesterol was not associated with a significant change in the percentage fat intake. The implication is therefore that the changes in alpha- and beta-cholesterol resulted from seasonal alterations in physical activity, or in outside exposure, rather than from fluctuations which have been shown to have occurred in the food intake. Further to this, it has been reported that alpha-cholesterol levels show little response to dietary manipulations, their concentration in the plasma being relatively constant although certain endocrine stimuli are known to alter their concentration (Olson and Vester, 1960).

The hypothesis that these changes in the lipoprotein cholesterol values might be related to changes in physical activity or 'outside exposure' (a term which would include the effects of cold stress and the influence of ultra-violet light) can be tested by examination of the levels in serum samples taken from the subjects on their return to base after sledging expeditions. Though the post-sledging results are out of place under the heading 'seasonal trends' it is convenient to examine them at this point due to the fact that

they shed some light on the seasonal changes in alpha- and beta-cholesterol.

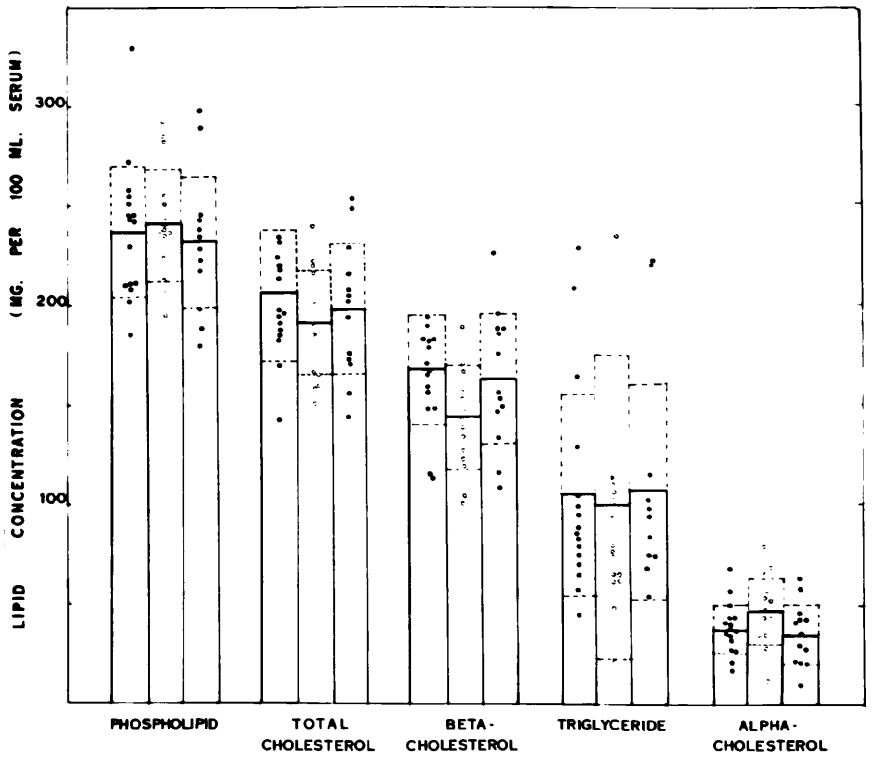
4. The Influence of Sledging on Serum Lipids

a) Background

The evidence that during dog sledging journeys there is a high energy expenditure and a high food intake, with especial emphasis on the intake of fat has been well documented. Recently Orr (1962) has shown that the total calorie intake may amount to above 5000 kcal/man/day, with a concomitant loss of body weight. Masterton, Lewis and Widdowson (1957) ascertained that the mean percentage of calories supplied by fat was 57%, while the total calorie intake ranged from 4520 to 4870 kcal/man/day. Sledging expeditions are invariably associated with some loss of body weight, which is regained rapidly on the return of the men to the base hut. The mean loss of weight after sledging of the Halley Bay subjects was 1.5 kg (see Appendix, Table 46). Thus sledging journeys were accompanied by an increase of food intake of approximately 1000 kcal, with a rise of 15% in the percentage of calories supplied by fat, and by a high energy expenditure of such magnitude that there was almost always some loss of body weight. It must be admitted that such a combination of circumstances is rare. It is clear that sledging journeys were also associated with increased outside exposure, and hence with a greater cold stress.

With the rise in the percentage of calories furnished by fat during sledging journeys the expected change in the beta-cholesterol would be an elevation, with in all probability no change in the alpha-levels. The prediction of the cholesterol level in subjects consuming a 57% fat calorie diet would be well above 300 mg %. Such a prediction

Figure 17. The influence of sledging journeys upon 5 serum lipid fractions. The apex of each block shows the mean concentration, and the interrupted lines represent ± 1 S.D. Each group of 3 blocks shows 1) levels in serum taken before the journey (closed circles) 2) levels in serum taken directly after sledging (open circles) and 3) levels in serum taken one month later (closed circles).



was made by Karvonen, Pekkarinen, Maitsala, and Rautanen (1961) in their studies on lumberjacks who were consuming a high fat diet.

b) Results

The serum lipid levels in subjects participating in sledging journeys are shown in Figure 17 in scattergram-cum-histogram form, and in Table 29. The diagram shows the results for five serum fractions, namely phospholipids, total cholesterol, with the alpha and beta-lipoprotein cholesterol fractions, and serum triglyceride concentrations.

Little change was indicated in the parameters in serum samples taken before and one month after sledging. These levels were therefore regarded as basal values in order to simplify the statistical analysis. From the meaned levels it appeared that the occasion differences might show significance for total and alpha- and beta-cholesterol values, and for the triglyceride levels. Analysis of variance and Scheffe's multiple comparison technique was therefore used for these parameters. The analysis was carried out on a single observation per subject, using subjects in whom observations were available on all three occasions. Where replicate results were available, the last result was used.

As was to be expected, subject differences were found to be significant for each of the responses of total, alpha- and beta-cholesterol, and for triglyceride concentrations. Only in the cases of alpha- and beta-cholesterol were significant occasion differences found. The levels of significance were 1% for alpha and 10% for beta-cholesterol. Scheffe considers that with his methods of analysis the 10% level can be said to indicate positive significance. The average effect for 'before' and for 'one month after' sledging, less the effect for 'immediately' after sledging was significantly different from zero at the 5% level for alpha-cholesterol and was significant at the 10% level for beta-cholesterol.

Table 29. The influence of sledging journeys on serum lipid levels. Demonstrated as means, with standard deviations.

Samples taken	Total Lipids	Total F.As.	Total Cholesterol	Phospho-lipids	C : P ratio	Tri-glycerides	β. Cholesterol	β. Cholesterol as % of total cholesterol	α. Cholesterol
Before sledging (16)	659.0 ±107.7	374.0 ±67.8	205.7 ±33.5	237.0 ±33.1	0.86 ±0.050	105.0 ±50.1	168.0 ±27.8	81.6 ±5.5	37.8 ±12.5
Directly after sledging (16)	638.7 ±108.6	364.3 ±73.4	191.4 ±25.3	239.6 ±28.1	0.79 ±0.061	99.1 ±75.9	144.4 ±25.1	75.5 ±8.5	47.2 ±16.9
One month after sledging (13)	646.1 ±101.0	371.2 ±67.9	198.5 ±32.4	231.6 ±33.4	0.85 ±0.058	106.5 ±53.5	163.3 ±32.4	81.9 ±7.7	35.2 ±15.0

c) Comments

The triglycerides and total cholesterol did not therefore show any response after sledging, though the total cholesterol did show a relatively small reduction in the mean level. However, the beta-cholesterol showed a significant fall of 21 mg % and the alpha-cholesterol showed a rise of 10 mg %. The fall of the total cholesterol was therefore dependent on that of the beta-cholesterol, and was diminished by the rise in the alpha-cholesterol concentration. A similar hypothesis to that which was proposed in the case of the seasonal changes over the year can be made: that the changes which occurred in the post-sledging alpha- and beta-cholesterol levels must have resulted from either outside exposure, or from the increased physical activity. From the increase which occurred in the fat intake, the expected result would have been a marked elevation of the beta-cholesterol, and it is seen that this did not occur.

The third factor which could have been associated with the sledging effects on the serum lipoproteins has already been enunciated, that of the loss of body weight. However, views are somewhat conflicting as to the causation of weight loss after sledging. Buskirk, Dee, Welch, Levy and Consolazio (1957) have obtained evidence in the field that the weight loss may be due to fluid imbalance rather than loss of tissue mass, and Orr (1962) has shown fairly conclusively that the depression in body weight was merely the result of a negative calorie balance.

If the subjects had not increased the percentage of fat in their diet, it is conceivable that the changes in total, alpha- and beta-cholesterol might have been more marked. During the sledging journeys it is possible that the following factors may have to some extent been responsible for the changes in blood lipids:-

1. The increase in physical activity.
2. The loss of body weight.
3. The outside exposure which was increased on the base level
    a) as ultra-violet radiation
    b) as a result of low ambient temperatures.

5. Discussion on Alpha- and Beta-cholesterol variations

The hypothesis that the changes in alpha- and beta-cholesterol may have been caused by the altering physical activity is probably the most acceptable of the three that have been suggested. The work of Nikkila and Konttinen (1962) on the effects of physical activity on post-prandial levels of fats in serum, indicated that there might be a relationship between serum lipoproteins and energy expenditure. The project largely concerned changes in non-esterified fatty acids, and serum triglyceride concentrations, but changes were also reported in both alpha- and beta-cholesterol levels. In the experiment, young soldiers were provided with a fatty meal and one half of the group were marched 16 kilometres, while the other half acted as a control and remained at rest. Total cholesterol and alpha- and beta-cholesterol were all depressed in serum taken after the return of the men to their camp. However, 2 hours later the beta-cholesterol levels were the same as in the previous estimation but the total and the alpha-cholesterol had risen. This in effect is similar to the changes in serum taken after the sledging journeys at Halley Bay.

During short periods of physical strain no marked changes in serum cholesterol have been observed (e.g. Beischer, 1956; Karvonen, Rautanen, Rikkonen and Kihlberg, 1958) or slight changes in the cholesterol

and phospholipid levels, the magnitude of which has been dependent on the degree of strain (e.g. Fahring and Wacker, 1932). During longer periods of physical activity Konttinen (1959) and Montoye, Huss, Brewer, Jones, Ohlson, Mahoney and Olson (1959) detected no correlation between the changes in serum lipids and the degree of physical activity. In studies on different groups of subjects it has often been reported that serum lipid levels were lower in physically active groups than in those performing sedentary work (Chailley-Bert, Labignette and Fabre-Chevalier, 1955; Keys, Anderson, Aresu, Biörck, Brock, Bronte-Stewart, Fidanza, Keys, Malmros, Poppi, Posteli, Swahn and del Vecchio, 1956).

The second factor mentioned above, that of the influence of weight reduction on serum lipid levels can largely be ignored. Though the serum changes occurred after sledging journeys which were associated with loss of body weight, similar changes occurred in the alpha- and beta- lipoprotein cholesterol at the beginning of the survey, at which time the body weight was gaining. It is therefore unlikely that the weight loss was per se the prime factor.

The third factor, the cold exposure, may have influenced the changes which occurred in serum lipids to some extent. From the data it is impossible to make a definite conclusion. That the seasonal changes in alpha- and beta-cholesterol levels might have resulted from variations in thyroid gland activity is possible. The rate of secretion of the thyroid hormone markedly influences the metabolism of lipids. Oliver and Boyd (1958) reported that l-tri-iodothyronine reduced total serum cholesterol and beta- lipoprotein, but actually elevated the alpha-fraction in the euthyroid state. On scanning the available literature only a few papers dealing with the influence of external temperatures

on human serum lipids were found. These are short-term studies and in some of them an increase in cholesterol at low temperatures (Kuhl, Beck, Gershberg, Street and Ralli,(1955) or a decrease at high temperatures (Marchioni and Ottenstein, 1931; Walinski and Bleish, 1939) has been observed and in others no clear changes (Ott, 1948).

It is apparent that there will be marked seasonal variation in ultra-violet radiation during an Antarctic year. It has been reported that ultraviolet radiation and sunlight bring about a decrease in cholesterol (Loeper and Degos, 1930; Altschul and Herman, 1953; Altschul, 1955; Plavsic, Strasser, Milutinovic and Nedvidek, 1958), whilst short-term exposure to strong sunlight and ultra-violet radiation causes a transient increase in cholesterol (Laureus , 1938). The seasonal changes found by Paloheimo (1961) in the group of convicts were not reflected in lipid levels of a group of policemen and therefore it seems unlikely that ultraviolet radiation plays more than a subsidiary role in the causation of seasonal changes.

The weight of the evidence is in favour of the changes in alpha- and beta-lipoprotein being due to activity variations and not due to alterations in cold or ultra-violet irradiation. The outside exposure in the summer months was reasonably high, and it was in these months that the sledging took place and when the outside temperatures were not excessively low.

It is of interest to point out two facts which have emerged from epidemiological surveys which have a bearing on the results of the present experiment. Keys et al (1958) observed that white females showed a higher level of alpha-lipoprotein cholesterol in their serum than males and Furman, Howard, Lakshmi and Norcia substantiated these findings in 1961.

Bloomberg, Lazarus, Mrost and Schneider (1958) demonstrated a markedly increased alpha-cholesterol level in the urban South African Bantu when compared with a local white population and noted that their beta-cholesterol was lower than the whites. However, the alpha-cholesterol level was much higher in the urbanised than in rural Bantu. Bloomberg and his associates explained this as being due to previous malnourishment of the rural group. However, it is relevant to point out that the serum samples which were drawn from the rural group were taken while the latter were under hospital treatment and is possible that under more active conditions the alpha-cholesterol of the rural and the urbanised groups might have been similar. The authors concluded also that the raised alpha-cholesterol might be related to the high excretion rate of oestrogen. The latter hormone has previously been shown to have a depressing effect upon serum beta-lipoprotein and an elevating effect upon alpha-lipoproteins. It is well known that the Bantu have a very low incidence of coronary thrombosis (Becker, 1946) and it is therefore arguable that any source that causes the lipoproteins to become quantitatively closer to the levels in the Bantu will have a beneficial effect upon ischaemic heart disease.

A recent report of a longitudinal survey in young American Army personnel (Clark, 1963) describes a progessive lowering of the high density lipoprotein fraction over a period of several years. At the same time the low density lipoproteins rose. The author discusses the changes in the high density lipoproteins at some length and concludes that there is evidence that the changes may result from parallel longitudinal changes in the ratio of androgen to oestrogen which occur in young men during earlier years of the third decade. There were no estimations of the urinary excretion rate of hormones to substantiate the statements.

Clark (1963) terminates by pointing out that it is a matter of considerable importance whether these changes are, in fact, produced by alteration of hormonal metabolism associated with normal maturation. He also observes that if diet or exercise is reponsible then a regimen of exercise and dietary control would be expected to reverse the unfavourable changes in the serum lipids. In the present experiment this has to some extent been answered. The period of intense exercise which ensued during the sledging expeditions influenced the serum lipoprotein levels to become closer to the normal levels.

The suggested relation of activity to beta- and alpha-cholesterol may be important. That beta-lipoprotein fractions are elevated in patients who have developed ischaemic heart disease and that there is a greater proportion of cholesterol in the beta-lipoprotein is well known (Jencks, Hyatt, Jetton, Mattingly and Durrum, 1956). Jencks et al found a depression of alpha-lipoproteins in patients with overt coronary disease. Ischaemic heart disease is less common and less severe amongst physically active people (Morris et al, 1953; Brown, Davidson, McKeown, and Whitfield, 1957; Spain and Braders, 1957; Morris and Crawford, 1958; Mathur, 1960) but the reasons for this have remained obscure. The findings in the case of the Halley Bay subjects may help to provide an answer. It follows that in order to gain a true picture of the correlation between cholesterol levels and the incidence of ischaemic heart disease, the lipoprotein fractions should be examined. It is clear that the relationship of physical activity to the alpha- and beta-lipoprotein levels can only be elucidated by further controlled experimentation.

In patients with ischaemic heart disease, the mean percentage of the total cholesterol in the beta-lipoprotein fraction has been found to be 86% (Bronte-Stewart, 1959). The Halley Bay mean beta-cholesterol

percentage was 81.8% and it is interesting that in the summer months the mean level lay in the region of 80% but, in the winter, the mean beta-cholesterol percentage rose to a maximum level of 85.4% which is uncomfortably near that shown by patients with ischaemic heart disease. Hence, though the change shown in alpha- and beta-cholesterol levels were small, these slight alterations very nearly placed the mean percentage beta-cholesterol within the zone expected in patients with ischaemic heart disease.

In the case of the sledging results, it is seen that the increased activity greatly improved the lipogram, but it is also interesting to note how soon the levels returned to their previous value. It can be construed that for physical activity to play a significant role in the prevention of coronary heart disease, then it must be upheld throughout the existence of the potential sufferer. Table 29 shows that sledging expeditions caused the mean C:P ratio to fall from 0.86 to 0.79 but that it rose to 0.85 4 weeks later. The percentage beta-cholesterol fell from 81.6 to 75.5% but rose to 81.9 a month later. It is obvious that the return to the previous base levels may have taken anything from 1 day to 27 days; no estimation can be made as to their rate of return to the pre-sledging level.

The important conclusions from the lipid data is that there is strong evidence that the lipoproteins are influenced in a beneficial way by physical activity. That the lipoproteins may be related to the aetiology of atheromatosis and of ischaemic heart disease has been discussed; it may be tentatively concluded from the present work that the triangular relationship between diet, beta-lipoprotein cholesterol and ischaemic heart disease may be extended to the four point relationship of diet, physical activity, beta-lipoproteins and ischaemic heart disease.

Figure 18. Serum triglyceride concentrations for 2 groups of subjects at monthly intervals. The shaded area shows the standard deviation for the 2 groups, and the central lines are the monthly means.

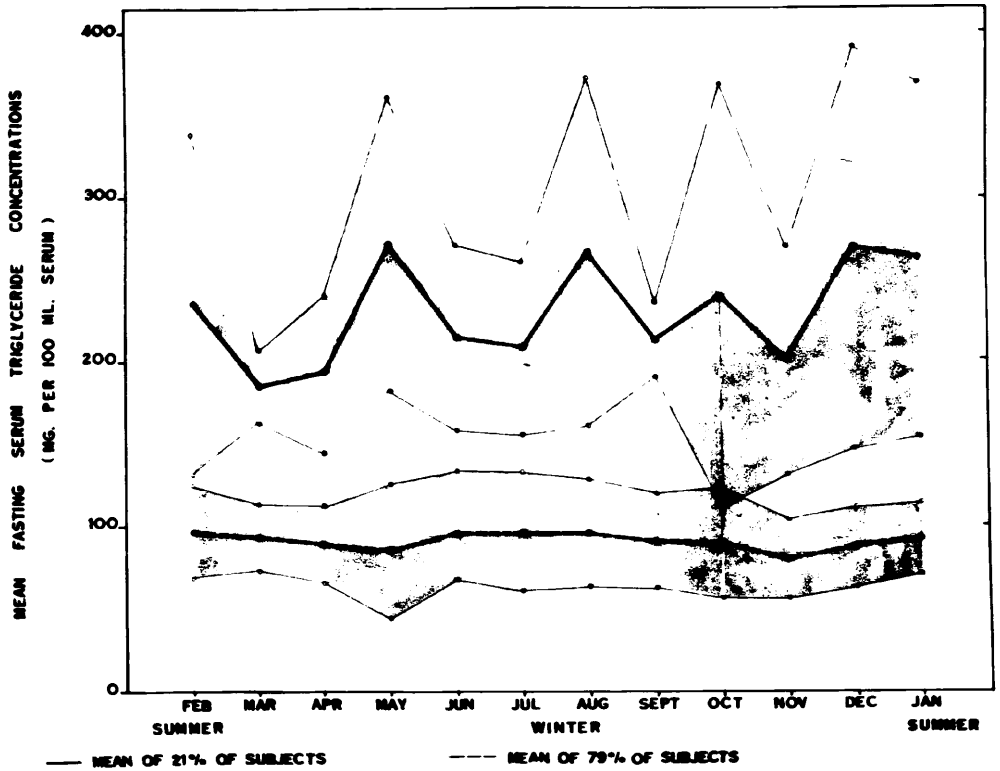


Table 30. The monthly mean changes in serum triglyceride values for the complete group with the monthly means for 5 subjects in a high triglyceride group, and 19 subjects in a low triglyceride group.

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
Means (all subjects)	125.6	105.6	110.6	125.7	122.0	117.8	131.2	115.2	122.2	106.4	125.1	127.9
S.D.	77.4	37.5	51.9	94.4	65.0	60.1	89.4	56.9	91.1	63.8	95.3	87.9
Mean (High T.G. group)	235.2	185.0	193.0	271.4	214.4	208.4	266.4	213.4	239.4	200.7	269.6	263.4
S.D.	103.0	22.7	48.1	89.3	56.6	53.3	105.8	23.3	128.8	69.3	122.5	109.5
Mean (low T.G. group)	96.8	93.6	89.4	85.2	96.0	96.5	95.6	90.5	89.6	79.4	87.1	92.3
S.D.	28.1	19.9	23.5	40.6	38.7	36.1	32.8	28.6	33.7	25.1	24.6	22.2
Grand Mean	120.8 ± 75.8											

6. Triglycerides

a) Results

The grand mean for serum triglyceride levels taking into account each serum estimation in the year was 120.8 ± 75.8 mg %. The monthly means and the standard deviations are set out in Table 30. Preliminary examination of the figures suggested the existence of two differing groups of subjects; those with high triglyceride levels (group I) and those with much lower levels (group II). The monthly means for the two groups have been demonstrated graphically in Figure 18 and Table 30. The mean individual serum triglyceride concentrations were found to be significantly different from each other at the 0.1% level by use of the 't' test for the two groups, and were shown to be significantly different by comparison in the analysis of variance for seasonal changes.

It is apparent from the monthly mean levels that the triglyceride concentrations for the whole group and the levels for the high and the low triglyceride groups demonstrated no trend over the year. This was substantiated by the result of the analysis of variance performed on the data, which indicated that a seasonal effect was non-significant. The analysis was performed on the triglyceride concentrations of all subjects with the exception of subjects SM, CJ, DED and MT. The months March and November were omitted from the analysis.

In Table 31 the triglyceride concentrations of the two groups with the high and low level have been compared with differences in other variables measured during the experiment. The triglyceride concentration for the high group was 231.3 ± 92.9 mg % and that for the low triglyceride group was 91.2 ± 31.4 mg %. From the Table it can be seen that the

Table 31. The mean triglyceride levels for the high and the low triglyceride groups, compared with differences in various parameters

	Mean Age (yr)	Body Wt (kg)	Mean max. gain in body wt. (kg)	Kg over-weight	Mean skinfold thickness (mm)	Total cal. intake	% fat intake (cal)	Fat: CHO ratio	B.P.		Pulse Rate	Total Chol-ester-ol	C:P ratio	Tri-glycer ides
									Systolic	Diastolic				
High T.G. Group	24.0	88.9	5.34	21.7	14.8	4055.0	41.7	0.900	116.2	79.5	62.8	220.8	0.92	231.3
Low T.G. Group	26.0	73.3	4.14	6.9	8.5	3522.0	39.5	0.814	107.3	71.4	56.1	193.0	0.85	91.2
Significance (t test)	NS	$p < 0.01$	NS	$p < 0.01$	$p < 0.01$	NS	NS	NS	$p < 0.05$	$p < 0.02$	$p < 0.01$	NS	NS	$p < 0.001$

high triglyceride subjects were significantly heavier and were more obese; also, that the diastolic and systolic blood pressures and the pulse rate were higher. The figures suggest that the subjects in the high triglyceride group showed a tendency to gain body weight to a greater extent than the subjects of the low triglyceride group and that they tended to consume a higher calorie intake, though the difference in the percentage of calories supplied by fat between the groups was minimal. The mean values for the cholesterol levels and the cholesterol: phospholipid ratios indicated that they might be elevated in the high triglyceride group, but they could not be proved to be significantly different by statistical methods.

b) Comments

During recent years, the glyceride level in serum has been studied in some detail and has been found to be often raised in patients who have suffered an overt myocardial infarction (Albrink and Man, 1959; Carlson, 1960; Antonis and Bersohn, 1960; Schrade, Boehle and Biegler, 1960). Albrink (1962) states that though the ominous significance of a very high cholesterol concentration cannot be denied, the cholesterol is more often normal than not in coronary artery disease. In 1961 Albrink, Meigs and Man reported that 82% of patients with coronary artery disease demonstrated triglyceride concentrations above 160 mg %, while between the ages of 40 and 69, 35% of normal subjects exceeded this limit.

Albrink and her colleagues (1961) went on to show that a serum triglyceride level of 160 mg % best separated a normal from a coronary population. Five of the Halley Bay subjects showed a mean triglyceride concentration above 160 mg %, while the remaining subjects all demonstrated

means well below this level. Albrink and her co-workers also stated that 5% of young males in the third decade had triglycerides above 160 mg %, so the percentage of men showing concentrations higher than this upper limit at Halley Bay does not show agreement, in that the percentage with elevated levels was 21%. Antonis and Bersohn (1960) in a study of serum triglycerides in differing ethnic groups proposed that the upper limit of a normal population should be 114 mg %. Their conclusion was based on triglyceride levels in the South African Bantu and thus was a good basis from which to define the bounds of normality. Using the upper limit as proposed by the South African workers, 37% of the Halley Bay subjects had elevated levels. Thus serial sampling at regular intervals of time appeared to demarcate a far greater population than normal showing elevation of their serum triglyceride concentrations. On the other hand, it might be accepted that the group was not a normal representative sample of the general population, or that the serum levels resulted from either a raised dietary fat intake, or that the samples were not truly fasting specimens. However, even if the latter observation were true, all samples of blood were taken at approximately standard times after the last meal and so were comparable on a time basis.

In a previous section, the percentage of calories supplied by fat, taking every dietary estimation into consideration, was stated to have been 39.8%. Adam, Best, Edholm, Goldsmith, Gordon, Lewis and Wolffe (1958) in a survey of recruits to the Army assessed the percentage of energy supplied by fat to be 38.6%. In the section which discussed the food intake results further evidence was brought forward to show that the fat intake of the Halley Bay subjects was not higher than intakes for young men in the United Kingdom. Thus the fat intake was not at variance with normal intakes, and does not supply an answer as

to the cause of the relatively large percentage of subjects with elevated serum triglycerides.

It is seen from Table 31 that the total calorie intake was not significantly higher in the group with raised triglyceride levels than in those subjects with the normal levels. In fact the calorie intake expressed per kilogram of body weight was 45.6 kcal/kg in group I and 48.1 kcal/kg in group II. The percentage of calories supplied by fat was not significantly different in the two groups, neither was the fat:carbohydrate ratio. There was no indication from the dietary intake figures that the five subjects in group I were prone to consume a greater proportion of their diet as fat than group II, though the fact that this could have been the case cannot be eliminated on the evidence of the somewhat sparse dietary intake results. · However, the relationship of diet and serum lipid levels will be discussed in some detail for each subject in the next section.

Table 31 shows that group I were heavier and more overweight, and tended to gain weight to a greater extent than subjects in group II. Group I subjects were more obese than group II subjects. All these facts indicate that the group I men had tended to gain weight in earlier years, with which the greater weight increase shown in the year by the group is in agreement. Albrink, Meigs and Granoff (1962) found that male factory workers who had gained more than 4.5 kg body weight since the age of 25 years had significantly higher serum triglyceride concentrations than men who had gained 4.5 or less since that age. It is tempting to suggest that the high levels of triglyceride concentrations in the group as a whole may have been related to the finding that almost all subjects on the base gained weight during the year. Finally, it can be seen that group I subjects had a slightly higher blood pressure and pulse rate than the subjects of group II.

It is interesting that a group of men in the third decade with elevated triglycerides should show a suggestion of raised blood pressure when compared with the group of normals.

The body of evidence is as yet not strong enough to allow that young men with raised triglyceride concentrations be instructed to change their dietary fat intake from one which contains a high proportion of saturated fatty acids to one in which the total fat intake is reduced and in which the amount of saturated fatty acids are lowered in favour of an increased intake of unsaturated fatty acids. Conclusions from the present study, and from the survey of Albrink et al (1962) on factory workers, imply that men in their twenties must not gain weight in excess. However, the views relating obesity to ischaemic heart disease are conflicting; reports of a strong correlation between coronary heart disease and obesity (Wilkins, Roberts and Morris, 1959) are countered by evidence that no such relation exists (Keys et al, 1954). Though the group I subjects had no family history of ischaemic heart disease, the occurrence of a slightly higher blood pressure fits into a premise that raised triglyceride concentrations in young men might be employed for the prediction of vascular disease in later life. Such a premise can only be confirmed by long term studies over many years as emulated by the Framlingham study initiated in 1948. However, the possibility exists that the elevation of the blood pressure of the group I subjects was due to the fact that they were on the average much heavier than group II subjects. There is known to be a positive correlation between blood pressure and body build.

The triglyceride, and similarly the total cholesterol and phospholipid concentrations, in no way follow the monthly trend of the total calorie, or the total fat intake. It has been shown on numerous occasions that alterations in these serum levels are dependent among

Table 32. Shows the influence of the seasons on food intake, with the percentage of total calories furnished by fat, the energy expenditure, and the change in body weight.

	TEMP. (°C.)		FOOD INTAKE	ENERGY	MEAN WT.
	max.	min.	(Kcal.)	EXPENDITURE	CHANGE
FEB. TO APRIL (daylight)	- 12·4	- 21·9	3850 38·9% fat	HIGH	+ 2·5 kg
MAY TO SEPT. (polar night)	- 23·3	- 34·9	3363 39·6% fat	REDUCED	+ 0·8 kg
OCT. TO JAN. (daylight)	- 6·0	- 12·9	3663 41·6% fat	INCREASED	- 0·4 kg

other things on dietary manipulations which involve the percentage of calories supplied by fat, or the fatty acid structure of the dietary fat. The fact that the serum levels do not follow the changes in the total calories or the fat calories is in agreement with many previous studies.

7. General Discussion

Several points emerge from the study of the lipid levels for seasonal changes. By far the most surprising result is for the most part the total absence of a seasonal change in the majority of lipid parameters. In conditions where the climate, physical activity, and dietary intake showed dramatic changes, the serum lipid levels showed minimal response. The changes which occurred in the alpha- and beta-cholesterol levels, although they showed some statistical significance, were small. It is apparent that man can adapt himself to the seasonal climatic changes that exist in Antarctica, which it can surely be said must be some of the most extreme in the world, and maintain his body weight constant by a delicate balance between the calorie intake and the energy expenditure.

It is relevant to the present work that a long term survey carried out by Calvy, Cady, Mufson, Nierman and Gertler (1963) investigated the effect of severe exercise on the serum lipid levels of young American Marine Corps trainees. These men were consuming an average of 4500 kcal/man/day and 45% of the total consumption was supplied by fat. At the same time their daily energy expenditure was high; they remained in iso-caloric balance throughout the greater part of the experiment. The serial assessments of total cholesterol and phospholipids indicated that the levels were in no way elevated. This compares with the effect of the severe physical exertion experienced

in sledging journeys, in which the serum levels tended to fall, possibly as a result of the exercise. Calvy et al (1963) also noted that the cholesterol:phospholipid ratio showed a fall over the 5 month period of the experiment and that the triglyceride concentrations demonstrated an increase towards the end of the survey. The authors conclude that the tendency for raised serum glyceride levels resulted from the highish intake of carbohydrate, both of which have previously been shown to be interrelated. They finalise by suggesting that exercise, if great enough to maintain an isocaloric balance, has a lipid lowering effect, and also an atherogenesis inhibiting effect. It is perhaps unfortunate that there was no control estimation made before and after the period of exercise.

Though it cannot be definitely stated at this point, it seems clear from the seasonal changes that the variations in the following serum levels were closely correlated; total cholesterol, beta-cholesterol and cholesterol esters. The phospholipid levels failed to show the depression in the last 6 months of the year which were evidenced on examination of the total cholesterol mean monthly level; further observations will be made concerning the relationship of these parameters in the section dealing with individual changes. The alpha-cholesterol level and therefore the alpha-lipoprotein concentration showed its own characteristic seasonal change. There was only a very slight overall fall during the year. When it is remembered that a greater percentage of phospholipid than cholesterol is contained in the alpha-lipoprotein (Figure 1), the disparity between the phospho-lipid and the cholesterol overall change is to some extent explained. More clearly, it seems likely that alpha-cholesterol concentrations would be reflected more closely in the serum phospholipid concentrations than in the total cholesterol levels, which correlate with changes

occurring in the beta-lipoprotein class.

It can be fairly stated that the serum triglyceride concentrations showed little correlation with any other lipid parameter which was measured. This last observation is of some interest; the greater proportion of triglyceride is carried in the chylomicrons or in the very low density end of the lipoprotein spectrum. From Figure 1 it may be seen that the chylomicrons contain 80% triglyceride by weight and thus variation in this portion of the lipid transport system will not be emulated in any of the other concentrations due to the fact that they are present in the chylomicrons in small quantities.

From the examination of the alpha- and beta-cholesterol changes both as monthly means over the year and in post sledging levels, it is tempting to conclude that there is a metabolic changeover of the high density lipoprotein to low density lipoprotein. A second hypothesis would be that both fractions were controlled by the same prime agent having opposing actions on the two classes of lipoprotein. The first suggestion is disproved to some extent by the fact that animal experiments using I^{131} labelled beta-lipoprotein have indicated that there is no metabolic turnover between alpha- and beta-lipoprotein (Gitlin, Cornwell, Nakasato, Oncley, Hughes and Janeway, 1958). The second suggestion could be explained either on the basis of an endocrine system such as the thyroid, or by the influence of the adrenal corticoids, perhaps secondary to changes in energy expenditure.

Finally, it is interesting to note that while the subjects were supposedly in a positive calorie balance over the months March and April, there was no evidence that any serum level was elevated, with the exception of the triglycerides. It could be construed therefore that the men were gaining weight due to increase of muscle mass rather than due to the storage of body fat. However, the increase in skinfold

thickness does not fit in with such a premise.

The effects of calorie excess on serum lipid levels have not been well defined. There has been no systematic study of variations in fat and carbohydrate calories, although profoundly different effects on the serum lipids might be expected. The study of Walker, Lawry, Love, Mann, Levine and Stare (1953) demonstrated in two men that excess calories over a short period caused striking increases in serum cholesterol and lipoprotein levels, even though the diet was low in fat. In an extension of the project Mann, Teel, Hayes, McNally and Bruno (1955) showed that these increases could be prevented if sufficient exercise were taken to dissipate the excess food calories. In a well controlled metabolic study of twenty physically healthy schizophrenic men Anderson, Lawler and Keys (1957) demonstrated that weight gains due to daily excess of 660 kcal caused significant elevations of total cholesterol. The levels reached a peak at five weeks, and were maintained unchanged for 15 more weeks despite continuing gains in weight.

From the results of the present survey it seems that positive calorie balance does not have an adverse effect upon serum lipoproteins as long as the energy expenditure is above a limiting value. The lipoproteins showed some derangement in the winter, when the men were virtually in isocaloric balance; however, the energy expenditure was comparatively low. Yudkin (1963) has said that in his opinion the differences in the incidence of ischaemic heart disease in different countries is determined by the surfeit of calories rather than the surfeit of fat. He points out that the surfeit comes from reduced physical activity and so in calorie need, without the necessary reduction in food consumption and so in calorie intake. From the present work it appears that positive or negative calorie balance are unrelated to the

distribution of the lipids in the lipoproteins; it seems that changes in physical activity per se does have an influence. Further longitudinal studies involving graded activities over a period of time might prove interesting. However, it must be remembered that the gain in weight might have been due to cellular material or to body fluids. This has already been discussed in Section II. If this were true, it seems fairly clear that it would not affect the levels of serum lipids in the same way as hypercaloric weight gain giving rise to obesity.

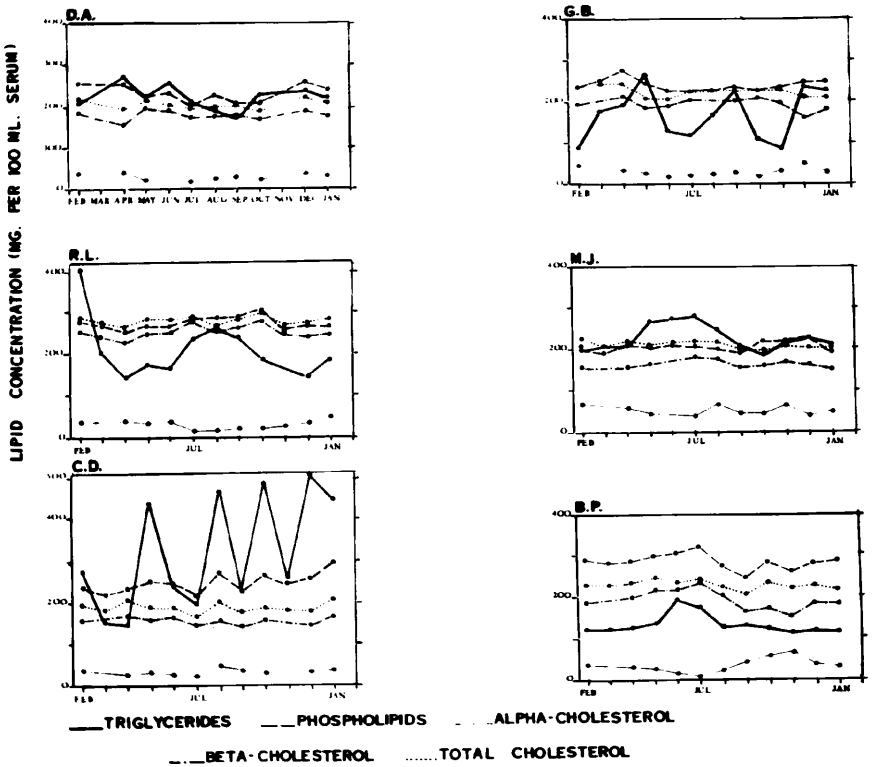
C. VARIATIONS BETWEEN MEN

The relationship of the serum lipid levels to seasonal changes in body weight, dietary intake, and energy expenditure has been discussed. Comment has been made concerning the influence of sledging expeditions and associated variables upon serum lipid levels . It is now proposed that the serum lipids will be examined for each person. The serum lipid derangements in coronary artery disease include elevation of serum cholesterol concentration and of the cholesterol:phospholipid ratio, a relative or absolute increase in low density or beta-lipoproteins, an increased triglyceride concentration, and exaggerated alimentary lipaemia (Albrink, 1962). Such may or may not be the case. Doubtless some years will elapse before a decision can be made for or against the serum lipid-ischaemic heart disease hypothesis.

1. Results

In the following, it will be shown that several of the subjects showed lipid abnormalities according to the data generally accepted as being the upper limit of the normal. It is not implied that the subjects will eventually succumb to ischaemic heart disease, it is only pointed

Figure 19. The individual monthly values for 5 serum lipid fractions, namely: triglycerides, phospholipids, total cholesterol and alpha- and beta-cholesterol. Levels shown for subjects DA, GB, RL, MF, CD and BP.



out that some of their lipid levels are within the range that would be expected in patients with coronary thrombosis.

The variations in the individual levels at monthly intervals have beendemonstrated in Figures 19, 20 and 21, and the average levels of various parameters assessed during the survey have been shown in Tables 33, 35 and 36.

DA - Age 23 years, Glaciologist

This subject showed a high mean triglyceride level of 221.8 mg % which showed little fluctuation when compared with subjects RL, CD and GB. His mean total cholesterol level was 203.4 mg% and his phospholipid level was 229.3 mg %. He demonstrated an elevated cholesterol:phospholipid ratio in the winter months when his activity was reduced.

Table 33 shows that the subject DA was heavier and more obese than most of the other men. His blood pressure and pulse rate were not raised. His caloric intake was not excessively high, but the percentage of calories supplied by fat was 43.7% which was above that of most of the other members of the expedition. Table 34 shows the data for body weight at specified times during the year. The maximum gain in weight after the first measurement was 5.2 kg. Judging by the mean skinfold thickness reading for the subject, he showed a tendency to gain weight in earlier life.

Thus, the cholesterol:phospholipid ratio was elevated, the subject showed a high beta-cholesterol and a low alpha-cholesterol and the triglyceride levels were consistently raised. There was no family history of coronary disease, and the subject showed no external sign of vascular disease.

Subject	DA	GB	RL	MJ	CD	BP
Age (yr)	23	26	22	27	28	23
Weight (kg)	84.34 ±1.68	101.63 ±1.20	97.71 ±1.38	71.08 ±1.95	89.55 ±1.80	69.88 ±1.30
Height (cm)	181.5	191.5	186.0	172.2	175.0	181.8
Total calorie intake	3987.8 ±536.5	4182.5 ±1035.3	5122.2 ±902.2	3634.4 ±564.8	3346.1 ±477.7	3496.4 ±792.5
% Protein	10.6	11.5	10.7	12.7	13.5	12.1
% Fat	43.7	39.3	45.6	38.1	41.9	36.0
% CHO	45.6	49.2	43.2	49.2	44.5	52.0
Mean skinfold thickness (mm)	12.5	29.5	12.4	7.4	12.2	7.0
B.P. (mm Hg) Systolic	107.6 ±11.0	113.7 ±11.6	124.5 ±9.3	112.5 ±8.7	122.8 ±7.9	106.8 ±12.8
Diastolic	70.0 ±6.7	79.6 ±8.0	85.0 ±7.7	78.0 ±4.0	85.0 ±7.9	69.5 ±10.1
Pulse rate (bts/min)	64.6 ±3.9	53.0 ±6.5	72.1 ±5.0	65.8 ±6.5	58.4 ±51	56.5 ±5.2
Total Lipid	775.4 ±59.9	752.0 ±65.9	913.5 ±78.3	766.3 ±47.6	854.6 ±155.7	749.1 ±74.6
Total fatty acids	482.3 ±37.3	454.3 ±66.0	531.0 ±55.9	472.6 ±31.5	574.9 ±140.3	461.0 ±34.5
Phospholipid	229.3 ±18.5	237.0 ±14.9	262.8 ±28.6	204.8 ±10.7	245.1 ±22.2	284.6 ±17.9
Total Cholesterol	203.4 ±11.5	222.2 ±13.7	280.3 ±10.5	209.5 ±8.9	188.5 ±11.8	227.6 ±10.9
Cholesterol esters	154.8 ±10.6	174.7 ±11.4	206.2 ±9.3	155.9 ± 9.2	133.4 ±11.7	167.3 ±9.6
Free Cholesterol	48.6 ±3.5	49.8 ±6.6	74.1 ±6.0	53.6 ±3.0	55.1 ±5.8	60.3 ±4.4
α-Cholesterol	25.2 ±8.7	26.8 ±9.8	28.4 ±10.1	50.1 ±10.6	32.2 ±6.7	34.7 ±16.1
β-Cholesterol	178.7 ±11.8	193.5 ±13.9	253.2 ±14.2	163.7 ±8.9	156.0 ±9.0	193.1 ±22.6
β-Cholesterol %	87.0 ±4.5	87.9 ±4.4	90.4 ±3.5	78.2 ±4.1	82.4 ±2.7	84.7 ±7.4
C:P ratio	0.89 ±0.06	0.93 ±0.06	1.02 ±0.05	1.01 ±0.01	0.77 ±0.06	0.79 ±0.03
Triglycerides	221.8 ±28.4	167.7 ±58.1	216.5 ±68.3	226.4 ±31.7	322.5 ±134.1	134.4 ±24.2

Table 33. The meaned individual results of the serum lipid levels (in mg/100 ml serum) and several other parameters assessed during the survey. The values for subjects DA GB, RL, MJ, CD and BP have been shown.

GB - Age 26 years, Aurora Observer

The triglycerides of the subject showed elevation and extreme fluctuation. The coefficient of variation for these levels was 34.6 which was higher than that of DA. From Figure 19 it is seen that five out of the twelve readings made could be accepted as lying within normal limits. This suggests that the serum samples with high triglyceride concentrations were non-fasting specimens. It is stressed, however, that the subject took all the precautions which were necessary, and it seems likely that the levels were as near fasting concentrations as those in the samples taken from other volunteers.

The total cholesterol level and the cholesterol:phospholipid ratio were both elevated. The beta-cholesterol percentage was also slightly elevated. The raised total cholesterol level predominantly due to a high beta-cholesterol concentration, with the raised cholesterol: phospholipid ratio suggested abnormality in the beta-lipoprotein; this finding, combined with the elevation of the triglycerides, points to an aberrent mechanism for the transport of the serum lipids, shown most markedly in triglyceride concentrations.

and

The subject was overweight/obese. He showed the highest skin thickness mean of all the subjects, namely 29.5 mm. Though his maximum gain in body weight was only 3.7 kg which was not great when compared with weight gains of many of the subjects, he demonstrated that in previous years he had considerably increased his body weight and become overweight.

The resting blood pressure was slightly raised at 113/79 mm Hg when compared with the mean level, whereas his pulse rate showed no elevation. There was no family history of ischaemic heart disease.

Table 34. Body weight changes of subjects stationed at Halley Bay

	Body Weights (kg)			
	February 1961	January 1962	Maximum	Minimum
DA	81.7	83.9	86.9	81.5
MB	69.9	72.5	75.6	69.9
GB	100.4	101.6	103.7	99.4
MBR	70.8	74.0	75.7	70.8
CD	87.2	92.8	92.8	87.2
ED	80.3	78.0	81.4	77.7
DE	73.1	74.2	77.5	71.4
DED	93.9	98.1	99.45	93.9
MJ	67.0	70.3	73.7	67.0
DJ	66.1	66.2	68.9	65.2
CJ	72.1	75.3	76.9	72.1
EJ	77.8	80.3	82.7	77.8
RL	95.4	101.3	101.3	95.4
SM	62.0	64.5	65.1	61.6
AM	66.3	68.8	69.2	66.3
GM	70.9	76.8	76.8	70.9
PN	72.4	77.8	78.7	72.4
BP	66.9	69.6	72.0	66.9
AP	68.2	69.5	73.4	68.2
JS	66.7	68.5	70.1	66.7
MS	61.9	64.4	64.4	61.9
GT	60.4	62.6	62.7	60.4
MT	77.6	80.6	82.8	77.6
ET	66.1	69.3	70.5	66.1
MTH	85.4	88.4	88.5	85.4
Mean body weights	74.4	77.2	78.8	74.2

RL - Age 22 years, Tractor Driver

For much of the year, subject RL's work was hard and involved a considerable amount of outside exposure. Whereas many members of the staff only periodically left the hut in the winter months to assist in digging out and manhauling of food and fuel, it was the duty of this subject to organise and assist at these times, and they were not infrequent. Thus his energy expenditure was probably the greatest of any subject during the year.

From Figure 19 it is seen that the February serum triglyceride concentration was raised above 400 mg % and it is questionable whether it can be accepted as a fasting level. In a survey of this nature where the cooperation of the subjects is of critical importance, to urge them to take low fat diets on the night before the venepuncture when appetites are keen, is difficult. However, the triglyceride levels remained elevated for the greater part of the year, and they showed a marked rise during the months July, August and September. The coefficient of variation of triglyceride levels for RL over the year was 31.6.

The mean total cholesterol level was 280.3 mg %, the mean cholesterol:phospholipid ratio was 1.02 and the beta-cholesterol expressed as a percentage of the total cholesterol was 90.4%. He thus showed a high beta-cholesterol level, and a reduced alpha-cholesterol concentration, which fits in with the elevated cholesterol:phospholipid ratio. A defect in the beta-lipoprotein fraction was well demonstrated in the subject, with the associated abnormal levels in the triglycerides, total cholesterol and cholesterol:phospholipid ratio.

Subject RL was overweight and obese. It is seen from Table 33 that his skinfold thickness measurement was high in comparison with

many of the subjects. His mean calorie intake was 5122 kcal/day, which showed a wide fluctuation (SD ± 902 kcal). The percentage of these calories supplied by fat was 45.6%. On this dietary intake the maximum gain in body weight was 5.9 kg. Thus the subject had a marked tendency to consume a high fat diet and to put on body weight. He also showed a slightly elevated blood pressure and pulse rate on comparison with the remaining subjects.

MJ - Aged 27 years, Geophysicist and Biologist

The triglyceride levels were raised through the greater portion of the year with a well marked winter elevation. The mean triglyceride concentration was 226.4 ± 31.7 mg %. The mean total cholesterol of 209.5 mg % was slightly raised above the mean level for the whole group and the cholesterol:phospholipid ratio was also raised at a mean of 1.01. However, the beta-cholesterol level as a percentage of total cholesterol was normal for this subject on comparison with the mean for the complete group. The elevated triglyceride concentration, with the raised cholesterol:phospholipid ratio is therefore difficult to explain on the basis of there being an abnormal transport of cholesterol and phospholipid by the beta-lipoprotein. However, the raised triglycerides might be explained either by an error in the deposition of chylomicrons (Kinsell, Michaels, Walker and Splitter, 1961) or by excessive concentration of the very low density lipoproteins (Havel and Gordon, 1960).

CD - Aged 22 years, Geophysicist

Like subject MJ, CD was a sedentary worker. His triglyceride levels showed a remarkable monthly fluctuation and were largely responsible

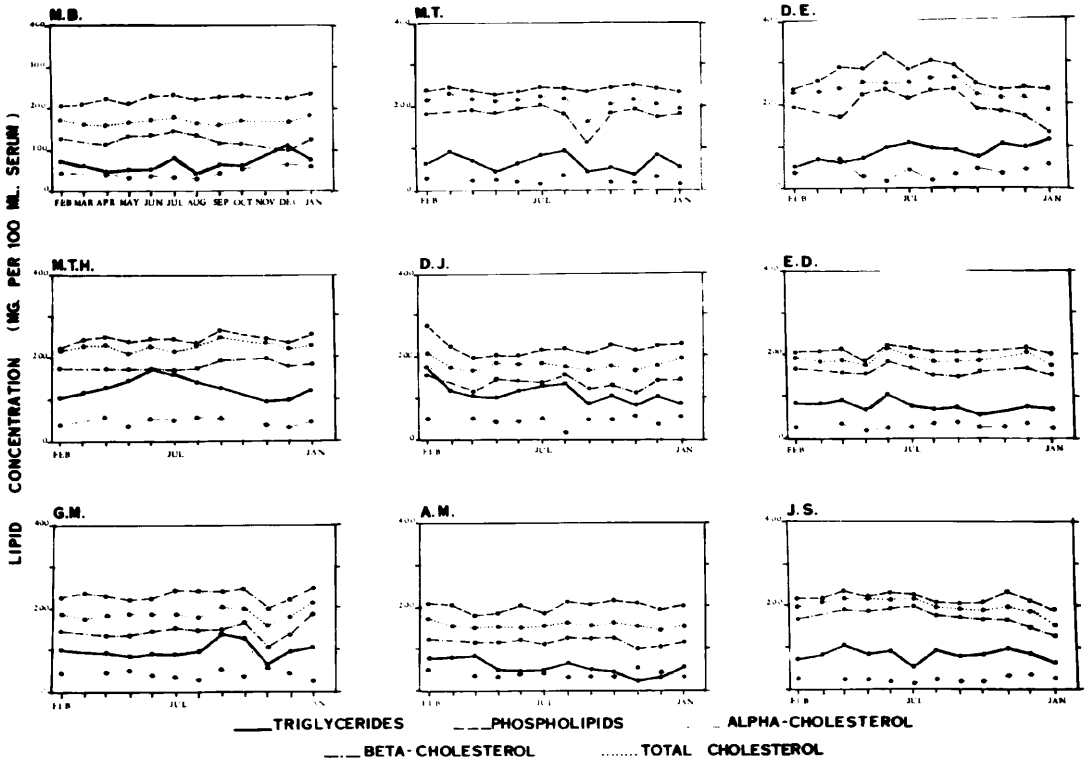
for the wide scatter of the five men of the high triglyceride group previously discussed. The mean triglyceride concentration was 322.5 ± 134.1 mg %, the coefficient of variation being high at 41.6. His mean total cholesterol level was 188.5 mg % and his cholesterol:phospholipid ratio was 0.77. It would appear that his lipoprotein was normal, the beta-cholesterol percentage being 82.4%. It is probable that the subject showed an aberrent mechanism in the serum for the clearance of chylomicrons, or the very low density lipoproteins. The marked degree of fluctuation shown would agree with this statement. The possibility also remains that there may have been a slow absorption from the gut, or again that the samples with an elevated triglyceride reading may not have been absolute fasting specimens. CD was overweight and also gained a maximum of 5.6 kg during the year.

The five subjects whose variables have been so far described allcomposed the group of subjects with high triglyceride levels (Group I). Three of the individuals showed obvious abnormality of the beta-lipoprotein system, but in subjects MJ and CD, the lipoproteins evidently were comparatively normal, though the triglyceride concentrations were markedly elevated. It has been suggested that in the latter two cases there might have been a triglyceridaemia due to an alimintary lipaemia with deficiency in the clearance of the triglycerides.

BP - Aged 23 years, Assistant Ionosphericist

Figure 19 shows that there were some interesting changes in the monthly lipogram of the subject. His triglyceride levels (mean 134.4 ± 24.2 mg %) were elevated and showed a rise in the months of June and July, which was not reflected in the total cholesterol level. However, the beta-cholesterol concentration (mean 193.1 ± 22.6) showed a distinct

Figure 20. The individual values for 5 serum lipid fractions, namely triglyceride, phospholipid, total cholesterol, and alpha- and beta-cholesterol, at monthly intervals. Demonstrated for subjects MB, MT, DE, MTH, DJ, ED, GM, AM, and JS.



winter elevation, while the alpha-cholesterol showed a fall. The rise in triglyceride levels during these months appears to have been associated with the rise in beta-lipoprotein cholesterol. However the fact that the serum phospholipid concentration increased in parallel with the beta-cholesterol winter rise is difficult to explain, and it suggests that the cholesterol:phospholipid ratio in the alpha-lipoprotein fraction may have been greater than 0.48 for the subject, the changes occurring in the phospholipid representing variations in the beta-lipoprotein portion. The cholesterol: phospholipid ratio in subject BP was 0.79 which therefore was not elevated.

MB - Aged 25 years, Ionosphericist

It is seen that the lipogram of the subject showed little fluctuation and Table 35 shows that the average values were within the bounds of normality.

MT - Aged 25 years, Meteorologist

The triglyceride levels of MT, mean 66.7 $\pm$ 18.7 mg %, remained below 100 mg % throughout the year. There was some fluctuation which demonstrated no trend (coefficient of variation 35.6), and which was not simulated in the other lipid parameters. The beta-cholesterol when expressed as a percentage of the total cholesterol was 87% and was slightly raised. This would account for the slight elevation in the cholesterol:phospholipid ratio, the mean of which was 0.87. The total and beta-cholesterol concentrations showed a sharp drop in the September post-sledging serum sample associated with a rise in the alpha-cholesterol level. The September serum phospholipids showed a minimal fall, due in the main to the rise in alpha-lipoprotein, a far greater proportion of phospholipid being contained in this lipoprotein class.

Subject	MB	MT	DE	MTH	DJ	ED	GM	AM	JS
Age (years)	25	25	27	23	25	25	25	23	25
Weight (kg)	72.36 ±1.67	80.10 ±1.96	75.13 ±1.65	87.08 ±1.10	66.52 ±0.94	79.80 ±1.05	74.41 ±1.92	68.10 ±0.74	68.82 ±0.98
Height (cm)	177.4	179.8	179.3	190.5	178.1	174.8	171.6	179.9	171.6
Total calorie intake	3113.3 ±263.8	2911.3 ±589.9	3589.5 ±586.7	4625.6 ±1483.3	3227.4 ±395.0	3628.6 ±238.8	2671.8 ±555.9	3784.0 ±651.9	3401.8 ±631.7
% Protein	12.3	13.9	11.3	10.1	11.2	13.0	13.2	11.1	12.5
% Fat	38.2	39.2	42.6	47.5	36.8	37.5	38.2	39.4	40.2
% CHO	49.4	46.9	46.1	42.2	51.9	49.5	48.6	49.5	47.3
Mean skin-fold thickness (mm)	9.9	11.3	7.5	9.5	7.1	8.8	11.5	6.1	7.8
B.P. (mm Hg) Systolic	102.3 ±8.0	114.2 ±11.3	-	115.0 ±11.0	100.3 ±6.6	109.5 ±9.9	99.5 ±11.2	110.0 ±11.2	105.8 ±12.4
Diastolic	71.4 ±6.7	74.6 ±9.7	-	69.5 ±7.6	69.3 ±7.1	71.4 ±7.1	72.3 ±10.5	79.5 ±9.9	68.7 ±9.4
Pulse rate (bts/min)	51.4 ±3.1	64.0 ±3.3	-	57.3 ±6.3	56.2 ±5.5	55.7 ±4.4	57.9 ±4.3	52.2 ±3.8	59.2 ±4.4
Total lipid	558.0 ±25.1	639.6 ±43.2	721.1 ±62.6	639.6 ±43.2	604.2 ±54.1	574.4 ±35.6	608.3 ±42.0	499.2 ±28.0	615.1 ±35.4
Total fatty acids	283.6 ±20.7	349.2 ±26.9	394.8 ±33.8	409.5 ±25.7	352.8 ±38.5	320.0 ±22.6	350.1 ±30.0	278.3 ±20.7	339.6 ±25.9
Phospholipid	223.1 ±9.6	240.7 ±6.1	270.5 ±28.6	244.1 ±11.2	215.7 ±19.1	206.0 ±9.7	227.8 ±12.9	199.5 ±11.0	212.6 ±11.8
Total Cholesterol	170.2 ±7.2	209.2 ±20.2	237.2 ±24.2	224.7 ±9.7	178.6 ±12.0	187.7 ±12.4	190.1 ±10.9	156.3 ±6.4	196.3 ±17.6
Cholesterol esters	124.8 ±7.5	159.6 ±24.8	172.5 ±19.8	163.0 ±7.3	135.1 ±10.7	141.5 ±10.1	140.4 ±11.8	118.8 ± 6.4	153.9 ±15.5
Free Cholesterol	45.5 ±3.7	47.1 ±5.6	64.6 ±9.9	61.7 ±4.2	43.5 ±3.2	46.3 ±4.6	49.7 ±4.4	37.5 ±3.4	43.3 ±4.2
α-Cholesterol	46.9 ±10.7	26.4 ±9.1	34.2 ±9.6	45.5 ±8.1	44.8 ±9.7	28.2 ±5.4	40.7 ±9.0	40.7 ±6.0	24.8 ±5.8
β-Cholesterol	129.9 ±13.6	183.1 ±21.8	199.3 ±32.2	178.6 ± 9.3	134.4 ±14.6	160.5 ±10.7	145.6 ±18.8	115.8 ± 8.0	171.3 ±20.8
β-Cholesterol %	72.4 ±6.3	87.3 ±5.6	83.6 ±6.6	77.5 ±5.2	74.9 ±5.8	85.7 ±2.2	77.6 ±5.5	74.1 ±3.7	87.5 ±3.7
C:P ratio	0.77 ±0.04	0.87 ±0.06	0.87 ±0.05	0.93 ±0.03	0.82 ±0.05	0.91 ±0.03	0.81 ±0.04	0.78 ±0.04	0.90 ±0.06
Triglycerides	67.7 ±17.5	66.7 ±18.7	86.7 ±17.7	127.4 ±24.2	110.6 ±24.2	77.6 ±11.7	99.0 ±18.4	56.6 ±18.4	93.1 ±14.4

Table 35. The meaned individual results of the serum lipid levels (in mg/100 ml serum) compared with the means of some other parameters assessed during the survey. Demonstrated with standard deviations for subjects MB, MT, DE, MTH, DJ, ED, GM, AM and JS.

Thus after a period of activity superimposed on a long term of inactivity, the lipoprotein pattern of the subjects was greatly improved.

Hence MT showed little abnormality of his serum lipid pattern. The effect of a sledging expedition in September, at which time outside temperatures were low, was dramatic; the main beneficial effect was shown in the cholesterol:phospholipid ratio, which remained at a lower level for the last months as compared with the first 7 months.

DE - Aged 27 years, Medical Officer

The mean triglyceride level was 86.7 $\pm$ 17.7 mg %, the coefficient of variation being 20.4. There was less fluctuation in triglyceride concentrations than in phospholipid and beta-cholesterol levels. Total cholesterol levels, mean 237.2 $\pm$ 24.2 mg % rose from the February level to that of the September reading, and then fell off in the remaining months. The beta-cholesterol levels demonstrated an elevation in the winter months, which was paralleled by the serum phospholipid levels; on the two occasions when this did not happen (April and January) the serum samples were taken after sledging expeditions. The alpha-cholesterol concentration showed a depression in the winter months.

A serum sample which was drawn in the United Kingdom before the expedition departed for the South demonstrated a cholesterol level of 250 mg %. In the first 3 months of the year, serum cholesterol concentrations were below this level, but from May to September, they exceeded this concentration.

MTH - Aged 23 years, Biologist

The triglyceride concentration of the subject was 127.4 $\pm$ 24.2 mg%. Figure 20 shows that the triglyceride levels showed a distinct winter rise,

and reached a maximum level of 174 mg % in June. The serum alpha-cholesterol levels showed a tendency to be elevated during the winter, while the beta-cholesterol (77.5% of the total cholesterol) showed minor variation. The cholesterol:phospholipid ratio was elevated, which, with the normal distribution of cholesterol in alpha- and beta-lipoproteins, is somewhat anomalous. The fat intake of the subject was the highest of all the volunteers, providing 47.5% of the total calorie consumption. However, on a food intake of 4625 kcal/day MTH gained a maximum of 3.0 kg in body weight, which was lower than the mean maximum body weight gained by all subjects. His mean skinfold thickness was 9.5 mm which was equivalent to the mean for the whole group.

DJ - Aged 25 years, Meteorologist

The subject demonstrated no abnormal lipid levels in his serum.

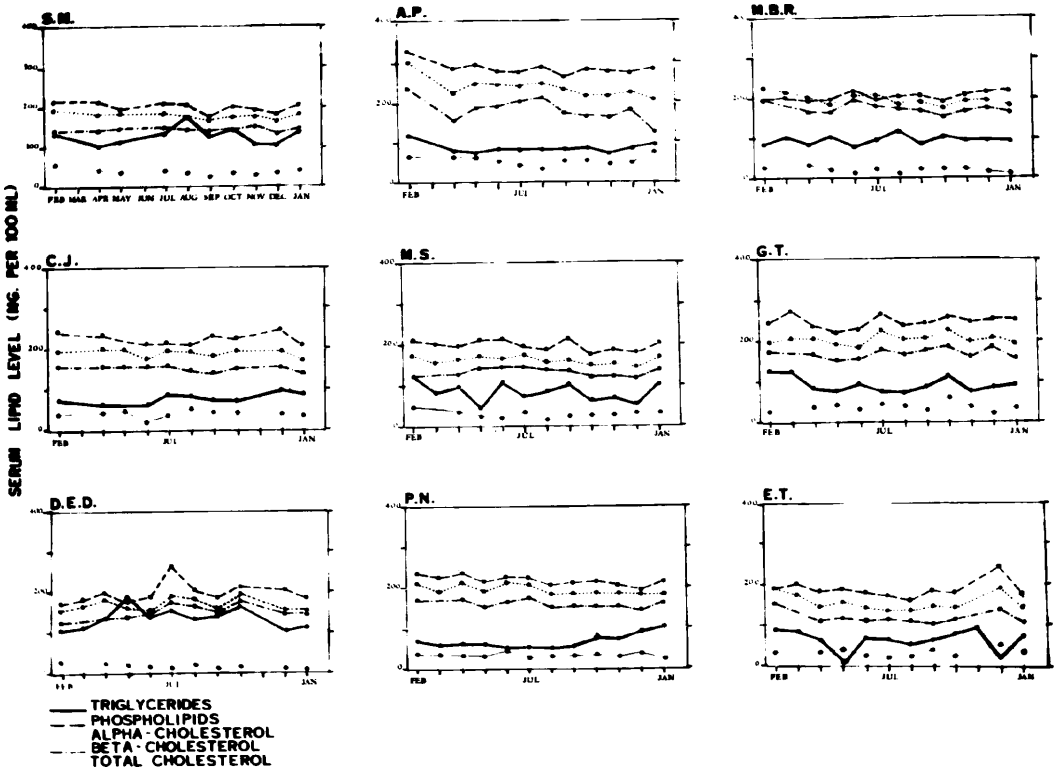
ED - Aged 25 years, Carpenter

The triglyceride levels fluctuated round a mean concentration of 77.6 mg %, the standard deviation being 11.7. The coefficient of variation of 15.0 was low when compared to the greater proportion of subjects. The beta-cholesterol concentration expressed as a percentage of the total cholesterol was slightly raised on the group average, at 85.7, as also was the cholesterol:phospholipid ratio of 0.91. This subject therefore demonstrated some abnormality of the lipoprotein composition.

GM - Aged 25 years, Cook

The total cholesterol level was 190.1 $\pm$ 10.9 mg % and the

Figure 21. The individual levels for 9 subjects over the year. Monthly variations in 5 serum lipid fractions are demonstrated. Shown for subjects SM, AP, MBR, CJ, MS, GT, DED, PN and ET.



cholesterol:phospholipid ratio was 0.81. The beta-cholesterol expressed as a percentage of the total cholesterol was 77.6%. GM therefore demonstrated no apparent abnormality in serum lipid levels. The fall in total cholesterol after a sledging journey (manhauling) amounted to 39 mg % and was coupled with a rise in alpha-cholesterol level of 23 mg %, while the beta-cholesterol was depressed by 62 mg %. The beneficial effect of strenuous activity upon serum lipid levels is beautifully demonstrated in this case.

AM,- Aged 23 years, Cook

The lipid levels of subject AM were all within the bounds of normality.

JS - Aged 25 years, Carpenter and General Assistant

The triglyceride levels of the subject remained at a constant level throughout the year, except for a sharp depression of 38 mg % in July. The mean serum triglyceride concentration was 93.1 mg %, and the standard deviation was $\pm$ 14.4 mg %. The total cholesterol level was 196.3 $\pm$ 17.6 mg %. Readings in the first 6 months were distinctly higher than those in the second half of the year. The cholesterol: phospholipid ratio was 0.90 and the percentage of total cholesterol linked with the beta-lipoprotein was 87.5%. Beta-cholesterol levels showed a rise in the winter months and the alpha-cholesterol (mean 24.8 $\pm$ 5.8 mg %) showed a tendency to fall. The elevated cholesterol phospholipid ratio fits in with the finding that the percentage of cholesterol in the beta-lipoprotein fraction was raised and suggests aberrent transport of cholesterol in this fraction.

SM - Aged 22 years, Physicist

The serum triglyceride concentration of subject SM was elevated

Subject	SM	AP	MBR	CJ	MS	GT	DED	PN	ET
Age (years)	22	34	26	30	25	26	25	27	22
Weight (kg)	63.53 ±1.16	71.38 ±1.48	73.69 ±1.39	74.45 ±1.32	63.52 ±0.72	61.73 ±0.62	96.81 ±1.80	77.23 ±1.67	68.86 ±1.30
Height (cm)	173.3	176.4	165.9	182.0	179.8	177.2	194.0	174.5	175.8
Total calorie intake	3588.9 ±777.2	3562.1 ±641.1	3000.0 ±572.7	3922.4 ±1136.0	3312.0 ±536.7	3357.3 ±334.7	5259.5 ±546.8	3604.4 ±432.4	2853.9 ±820.4
% Protein	15.1	10.8	10.7	11.4	12.2	13.4	10.6	13.0	10.3
% Fat	36.6	40.0	38.7	37.9	40.3	42.5	34.3	45.3	39.4
% CHO	48.3	49.2	50.6	50.6	47.5	44.1	55.0	41.7	50.3
Mean skin-fold thickness (mm)	6.3	6.9	11.3	6.5	7.3	6.3	8.0	13.2	9.6
B.P. (mm Hg) Systolic	99.0 ±14.3	106.5 ±10.3	103.0 ±6.8	106.0 ±13.7	101.7 ±11.1	118.2 ±6.2	105.0 ±9.5	101.7 ±13.1	127.0 ±6.8
Diastolic	58.1 ±12.3	72.5 ± 8.4	70.0 ± 6.7	74.5 ± 6.9	67.9 ±7.5	80.0 ±5.6	70.0 ± 9.5	64.2 ± 6.4	81.0 ±9.2
Pulse rate (bts/min)	56.6 ±4.1	49.2 ±4.3	55.9 ±6.9	52.4 ±2.3	58.1 ±5.8	56.9 ±4.3	54.9 ±3.3	55.8 ±5.1	60.2 ±5.0
Total lipids	604.5 ±33.5	739.0 ±58.1	603.3 ±18.2	599.4 ±26.7	533.5 ±30.6	658.7 ±37.9	596.8 ±47.5	591.3 ±26.7	492.2 ±50.8
Total fatty acids	358.1 ±25.3	412.5 ±30.8	341.0 ±34.1	334.4 ±14.4	304.0 ±30.8	372.6 ±25.6	357.5 ±27.7	324.9 ±14.8	276.2 ±29.4
Phospholipid	198.0 ±11.0	283.6 ±16.6	203.4 ±8.0	227.0 ±14.5	197.8 ±11.3	246.9 ±15.2	200.4 ±23.8	217.5 ±12.5	189.8 ±19.4
Total Cholesterol	179.8 ±8.1	236.0 ±25.4	196.5 ±13.4	190.9 ±10.3	160.6 ±9.3	204.2 ±12.0	170.0 ±15.8	195.5 ±12.2	155.2 ±20.9
Cholesterol esters	137.8 ±8.7	184.5 ±22.5	150.5 ±12.4	142.5 ±8.9	122.0 ±10.7	156.3 ±8.5	131.8 ±11.6	147.6 ±10.7	119.9 ±19.6
Free Cholesterol	42.0 ±2.9	51.5 ±4.1	46.0 ±3.7	48.4 ±3.9	38.6 ±4.9	48.0 ±5.7	38.2 ±5.7	39.8 ±2.6	35.3 ±4.8
α-Cholesterol	37.8 ±7.7	54.7 ±12.7	21.0 ±6.6	39.5 ±8.3	27.3 ±8.9	36.6 ±10.6	17.5 ±5.4	35.1 ±5.2	33.8 ±8.4
β-Cholesterol	142.0 ±4.8	180.4 ±27.3	174.2 ±12.5	152.4 ±7.3	133.6 ±10.9	169.6 ±11.9	155.3 ±17.5	161.2 ±9.4	125.3 ±20.3
β-Cholesterol %	70.1 ±3.0	76.3 ±6.0	88.9 ±3.7	79.5 ±2.0	83.2 ±5.8	83.4 ±3.3	89.0 ±3.6	82.3 ±2.2	78.0 ±4.0
C:P. ratio	± 0.91 ± 0.04	± 0.83 ± 0.06	± 0.95 ± 0.07	± 0.83 ± 0.04	± 0.82 ± 0.04	± 0.82 ± 0.04	0.88 ± 0.05	0.89 ± 0.02	0.82 ± 0.07
Triglycerides	126.6 ±21.1	84.2 ±8.6	95.4 ±9.7	79.5 ±10.5	82.8 ±23.0	92.9 ±18.6	135.9 ±24.7	69.5 ±15.4	59.8 ±24.1

Table 36. The meaned individual results of the serum lipid levels (in mg/100 mg serum) compared with the means of some other parameters assessed during the survey. Demonstrated with standard deviations for subjects SM, AP, MBR, CJ, MS, GT, DED, PN and ET.

over the upper level of normal proposed by Antonis and Bersohn (1960) at 126.6 mg %, the standard deviation being ± 21.1 mg %. Four out of the ten triglyceride estimations for the subject were lower than 114 mg % but were all greater than 100 mg %. The total cholesterol levels of the subject showed a mean of 198.0 ± 11.0 mg %, and the cholesterol:phospholipid ratio was elevated at 0.91. However, the percentage of cholesterol in the beta-lipoprotein fraction which was 70.1%, indicated that the lipoprotein transport mechanism was well regulated. Thus the anomaly of an elevated cholesterol:phospholipid ratio combined with the normal distribution of cholesterol in the lipoprotein classes recurs.

AP - Aged 34 years, Meteorologist

The monthly triglyceride concentrations (Figure 21) for AP were remarkable for their lack of fluctuation. The mean triglyceride level was 84.2 mg % and the standard deviation was 8.6. The mean total cholesterol level was 236.0 ± 25.4 mg % and the cholesterol: phospholipid ratio was 0.83. The cause of the high standard deviation for total cholesterol was the elevated level of the first month. The February total cholesterol was 300 mg % and the April reading was 222 mg %; thus, the fall amounted to 78 mg %. The depression in the beta-cholesterol level was of the same order as that of the total cholesterol. This marked fall, which also occurred to a lesser extent in the phospholipid fraction, ensued after a strenuous period of dog sledging in low temperatures which lasted for 5 weeks. It can be seen that the alpha-cholesterol concentration demonstrated no change and accounts for the smaller amplitude of the phospholipid depression. The effect of the strenuous journey is well shown in the cholesterol: phospholipid ratio, which fell from 0.91 in February to 0.77 in April.

The mean total cholesterol level of 236.0 mg % was higher than the average for the group. However, this subject was aged 34 years, which seemingly would account for the elevated concentration. The alpha-cholesterol level for AP was the highest of all subjects.

MBR - Aged 26 years, Radar Mechanic

The triglyceride levels for subject MBR fluctuated around the mean of 95.4 mg %. There was no obvious trend shown and the coefficient of variation was low at 10.2. The mean cholesterol level was 196.5 $\pm$ 13.4 mg %, 88.9% of which was transported in the beta-lipoprotein class. The cholesterol:phospholipid ratio was 0.95 which was high in comparison with normal values. The mean alpha-cholesterol remained at a low level for the greater part of the year. Thus MBR, although demonstrating no outright elevation of lipid levels, produced evidence that the relative amount of cholesterol and phospholipid in the beta-lipoprotein fraction was raised thus increasing the ratio of cholesterol:phospholipid.

CJ - Aged 30 years, General Assistant; MS - Aged 25 years Meteorologist; GT - Aged 26 years, Diesel Mechanic.

The above three subjects showed no changes of interest their lipograms, or in their meaned results.

DED -Aged 25 years, Carpenter

The serum triglyceride concentration was 135.9 $\pm$ 24.7 mg %. This concentration is above that suggested as being the upper limit of normal by Antonis and Bersohn (1960). The elevated percentage of cholesterol in the beta-lipoprotein (89%) confirms the evidence for an aberrent transport mechanism in the beta-lipoprotein fraction, which was

Table 37. Coefficients of correlation based on measured values shown in Tables 33, 35 and 36.

Correlation tested between:			Correlation coefficient (r)	Significance
Total Cholesterol	and	Age in years	0.19	NS
"	"	Total calories	0.39	NS
"	"	Fat calories	0.53	$p<0.01$
"	"	CHO calories	0.18	NS
"	"	Pr calories	0.35	NS
"	"	% intake of fat	0.49	$p<0.05$
"	"	% intake of CHO	-0.37	NS
"	"	% intake of Pr	0.17	NS
"	"	Serum TG levels	0.37	NS
"	"	Total lipids	0.80	$p<0.001$
"	"	Beta-cholesterol	0.95	$p<0.001$
"	"	C:P ratio	0.52	$p<0.02$
"	"	Skinfold thickness	0.39	NS
"	"	Max. wt. gain	0.19	NS
"	"	kcal/kg body wt.	0.27	NS
"	"	Diastolic B.P.	0.25	NS
"	"	Systolic B.P.	0.32	NS
Triglycerides	"	Age in years	-0.32	NS
"	"	Total calories	0.35	NS
"	"	Fat calories	0.36	NS
"	"	CHO calories	0.22	NS
"	"	Pr calories	0.12	NS
"	"	% intake of fat	0.25	NS
"	"	% intake of CHO	-0.22	NS
"	"	% intake of pr	0.03	NS
"	"	Skinfold thickness	0.31	NS
"	"	Max. gain in body wt.	0.39	NS
"	"	kcal/kg body wt.	-0.07	NS
"	"	Diastolic B.P.	0.39	NS
"	"	Systolic B.P.	0.39	NS
Beta-cholesterol	"	Fat calories	0.58	$p<0.01$
"	"	% intake of fat	0.56	$p<0.01$
Alpha-cholesterol %	"	% intake of fat	0.47	$p<0.05$
C:P ratio	"	% intake of fat	0.78	$p<0.001$
C:P ratio	"	Fat calories	0.53	$p<0.01$
Alpha-cholesterol	"	% intake of fat	0.12	NS
Phospholipids	"	% intake of fat	0.45	$p<0.05$
C:P ratio	"	Beta-cholesterol %	0.41	NS
Phospholipids	"	Alpha-cholesterol	0.22	NS

in the first place suggested by the raised triglyceride concentrations. The slight elevation in the cholesterol:phospholipid ratio lends support to the suggested derangement in the beta-cholesterol. It is clear also that the raised triglyceride concentration in the subject was due to augmentation of the beta-lipoprotein class and was not due to an elevation of the very low density chylomicrons. The alpha-cholesterol level of 17.5 mg % of subject DED was the lowest of all means demonstrated by members of the group. There was a downward trend of alpha-cholesterol from February 1961 to January 1962, which cannot be explained. The total cholesterol mean level was 170 $\pm$ 15.8 mg %. Thus, though the distribution of cholesterol in the alpha- and beta- lipoprotein fraction was distinctly abnormal, there was no elevation of the total cholesterol.

PN - Aged 27 years, Radar Mechanic, and ET - aged 22 years, Meteorologist

The above two subjects demonstrated no points of interest in their lipid levels.

The correlations between some pairs of parameters demonstrated in Tables 33, 35 and 36 have been calculated and shown in Table 37.

As was to be expected there was no correlation between the mean levels of serum triglycerides and total cholesterol concentrations. Total cholesterol, beta-cholesterol, beta-cholesterol %, phospholipid and cholesterol:phospholipid ratio all showed a positive correlation with the percentage of the total calories supplied by fat. Total cholesterol, beta-cholesterol and the C:P ratio also showed good correlation ($p < 0.01$) with the dietary fat intake expressed as calories. Total cholesterol showed no significant correlation with the total calorie intake and the calorie intake of protein and carbohydrate. The correlation of serum triglycerides and alpha-cholesterol with the dietary fat percentage were

non-significant. Furthermore, serum triglycerides and cholesterol levels showed no correlation with the percentage intakes of protein and carbohydrate.

From Table 37 it may be seen that the correlations between serum lipids and the skinfold thickness, with the maximum amount of weight gained from the first evaluation at the beginning of the year, were again non-significant.

There was no significant relationship between the cholesterol and triglyceride concentrations and the diastolic and systolic blood pressure. The correlations between total cholesterol and beta-cholesterol, total lipids and cholesterol:phospholipid ratio were all significant.

Finally, the correlation between the cholesterol:phospholipid ratio and the beta-cholesterol percentage was just non-significant.

2. Discussion

The standard deviation for the phospholipid concentrations ranged between 6.1 and 28.6 mg %, the mean individual standard deviation being 15.2 mg %. The highest level amounts to 329 mg % (Subject AP, February) and the lowest level recorded was 166 mg % (Subject ET, June). Paloheimo (1961) reported a mean individual standard deviation of 17.8 mg % in a group of policemen, while in a convict population the mean individual standard deviation was 19.3 mg %, The range for the former group was between 7 and 39 mg % and for the latter group it lay between 9 and 31 mg %.

In the case of the total cholesterol levels the standard deviation lay between a minimum of 6.4 mg % and a maximum of 25.4 mg %, the average being 13.1 mg %. The maximum recorded concentration was 300 mg % (AP, February) and the minimum level 142 mg % in serum of subject ET in August. There have been several reports concerning the

variation of serial measurements of total cholesterol levels over varying periods of time, the majority dealing with studies of fluctuations through some months or a year.

Luden (1917) determined his own cholesterol level for 7 months, finding it to be fairly constant. In different papers the range of variation of the cholesterol level has been from 4 to 27% (e.g. Turner and Steiner, 1939; Gordon and Brock, 1958; Thompson, Abraham, Elias and Scott, 1959) and expressed in mg/100 ml from 5 to 24 mg/100 ml (Man and Gildea, 1936). A variation of as much as 125 mg % was reported by Friedman et al (1958).

Schube (1936) reported individual differences between 19 and 73 mg % in the range of variation of cholesterol values. Morrison, Gonzales and Hall (1949), Josephson and Dalberg (1952) and Watkin, Lawry, Mann and Halperin (1954) observed deviations of about 10 to 13 mg/100 ml from the mean level of cholesterol. In thirty subjects studied by Gordon and Brock (1958) the intra-individual range was from 26 to 96 mg %, the average for the group being 45 mg %. This implies a standard deviation of the mean of 7 to 27 mg %. A similar range was noted by Thomas and Eisenberg (1959), who regarded a standard deviation of 25 mg % as the upper limit for the normal variation in cholesterol. In a series studied by Thompson et al (1959) one-third showed a variation exceeding 40 mg %. In a series of convicts studied by Thomas, Hollijes and Eisenberg (1961) the individual standard deviation during one year ranged between 10 and 49 mg % and the difference between the highest and lowest values noted in these subjects ranged between 38 and 150 mg %.

The variations in cholesterol level observed over periods of several years (e.g. Pucher, Griffith, Brownwell, Klein and Carmer, 1934; Man and Gildea, 1937; Sperry, 1937; Peters and Man, 1943; Steiner and Domanski, 1943; Sperry and Webb, 1950b; Paterson and Derrick, 1957;

Thomas and Eisenberg, 1957; Groover, Jerrigan and Martin, 1960) have been of the same magnitude as the variations noted during shorter periods, which have been discussed in the foregoing. Expressed in percentage, the variations in cholesterol have ranged between 1 and 31% of the mean. In Sperry's (1937) series, seventeen subjects out of twentyfive exhibited a variation of less than 6% of the mean value, whilst the maximum variation was 12%. Paterson and Derrick (1957) noted a variation of 11% during a period of 4 years.

Peters and Man (1943) reported individual variations in the level of cholesterol ranging between 1 and 68 mg % of the mean during a period of four years. In a study by Man and Gildea (1937) the maximum variation was 66 mg %. Thomas and Eisenberg (1957) and Groover et al (1960) regarded a deviation of 25 mg % from the mean value as normal. In the last mentioned paper a variation of 50 mg % was reported in thirtyseven out of one hundred and seventeen.

Irrespective of the length of the test period, greater fluctuations have been observed in subjects with high cholesterol values than in those showing low cholesterol values (e.g. Chandler, Lawry, Potee and Man, 1953; Thomas and Eisenberg, 1957; Peterson, Wilcox, Haley and Keith, 1960). There is concensus of opinion that repeated determinations at fixed intervals are required for the true evaluation of individual cholesterol levels (e.g. Thompson et al, 1959; Groover et al, 1960; Segall and Neufeld, 1960) and the results of the present survey suggested a similar conclusion.

The positive correlations between cholesterol levels and the total fat intake, and also the percentage of calories supplied by fat are of some interest. Gordon (1959) and Keys (1957) have presented epidemiological evidence which supports the theory that the major dietary

factor which affects the serum cholesterol levels is the total fat, or the proportion of calories supplied by fat. Thus they demonstrated a linear relationship between the mean serum cholesterol level and the dietary fat calorie percentage.

Recently Morris, Marr, Heady, Mills and Pilkington (1963) have published the results of a survey of diet and plasma cholesterol in ninetynine bank men. These workers could not establish a correlation between the dietary intake of protein, fat or carbohydrate and serum cholesterol. They attempted to correlate a great number of food categories with serum lipids with negative results. However, it is of interest that Morris and his colleagues report that there was a negative correlation between the carbohydrate intake and cholesterol levels, which was non-significant. The present work therefore shows some agreement in that the percentage intake of carbohydrate showed a negative non-significant correlation with serum total cholesterol, which was also demonstrated between the triglycerides and the carbohydrate intake.

The reason why Morris et al could not establish significance in their much larger group of subjects is of interest and may have been due to the fact that a) they did not establish the true cholesterol levels (i.e. that the number of estimations per person was not enough) or b) the assessments of dietary intake were not representative, again due to the fact that the surveys were for the most part carried out over a week twice during a year. The Halley Bay survey has the advantage that as the assessments were made at regular intervals over the year, seasonal changes were taken into account.

Other investigators have failed to substantiate the pre-eminence of the total dietary fat. In the first instance, the effects

of low fat diets were often disappointing, very severe restriction of dietary fat being necessary to produce a moderate decrease in serum cholesterol levels. For example, in a group of thirteen men Anderson and Keys (1953) achieved a fall of only 21 mg % by changing the fat content of the diet from 140 to 70 mg % (i.e. 37% to 18.5% fat calories) for 4 weeks. Further, the bias has been in favour of the degree of saturation of the dietary fat playing the major role (Bronte-Stewart, 1958) or vegetable versus animal fats.

The correlation of the individual serum cholesterol levels with dietary fat found in the present survey suggests that the personal cholesterol concentration may be dependent upon the habitual dietary intake of fat. With the observation of Anderson and Keys (1953) it cannot be dogmatically stated that small changes in dietary fat intake will influence serum cholesterol levels.

It has already been pointed out that differences in serum cholesterol levels correlate closely with the degree of unsaturation of the dietary fat. The Halley Bay fat intake was for the most part saturated. No oils were employed in the cooking. A rough estimation would place the percentage of saturated fat at about 80%. It is very probable that this did not vary over the year. The possibility exists that this is the reason why a correlation has been established between serum lipids and dietary fat intake in the Halley Bay results. The periodic changes in the ratio of saturated to unsaturated fats may influence the cholesterol levels and disguise the significance of correlations looked for in other surveys.

The absence of a winter fall in cholesterol levels, which would have been expected in view of the above correlation, is difficult to explain. It can be hypothesised that the expected winter rise which has

been shown to occur by other workers was balanced by the fall which occurred in the calorie intake of fat and that the final effect was only a very slight winter elevation. If the winter depression had not occurred in dietary fat it is conceivable that there would have been an elevation of the cholesterol levels during this time.

That the serum triglycerides showed no significant correlation with the fat intake was to be expected. The triglycerides show a much higher degree of variation than the cholesterol and phospholipid concentrations. It has already been pointed out that the triglycerides probably represent the very low density, or the chylomicron class of serum lipoproteins, in contrast to which the cholesterol levels indicated the state of the low density lipoproteins (Sf 0 - 12). It is rarely possible to be sure whether the triglyceride concentrations represent the very low density, or the chylomicron class. In many subjects the triglyceride level can represent a prolonged alimentary lipaemia.

It is seen from Table 37 that triglycerides and total cholesterol were not significantly correlated with the percentage intake of dietary protein and carbohydrate. It has been proposed that the raised serum lipid levels in western populations might be due to the elevated intakes of carbohydrate, or reduced intakes of protein just as much as to elevated intakes of dietary fat (Yudkin, 1957 and 1963). The lack of correlation shown in the intakes of carbohydrate and protein and the serum concentration of cholesterol would not support such a premise.

The absence of correlation between the age in years and cholesterol levels again was the expected result, the scatter of ages being narrow. Tanner (1951) reported positive correlation between the skinfold thickness and cholesterol concentrations, whilst Keys (1954) and

been shown to occur by other workers was balanced by the fall which occurred in the calorie intake of fat and that the final effect was only a very slight winter elevation. If the winter depression had not occurred in dietary fat it is conceivable that there would have been an elevation of the cholesterol levels during this time.

That the serum triglycerides showed no significant correlation with the fat intake was to be expected. The triglycerides show a much higher degree of variation than the cholesterol and phospholipid concentrations. It has already been pointed out that the triglycerides probably represent the very low density, or the chylomicron class of serum lipoproteins, in contrast to which the cholesterol levels indicated the state of the low density lipoproteins (Sf 0 - 12). It is rarely possible to be sure whether the triglyceride concentrations represent the very low density, or the chylomicron class. In many subjects the triglyceride level can represent a prolonged alimentary lipaemia.

It is seen from Table 37 that triglycerides and total cholesterol were not significantly correlated with the percentage intake of dietary protein and carbohydrate. It has been proposed that the raised serum lipid levels in western populations might be due to the elevated intakes of carbohydrate, or reduced intakes of protein just as much as to elevated intakes of dietary fat (Yudkin, 1957 and 1963). The lack of correlation shown in the intakes of carbohydrate and protein and the serum concentration of cholesterol would not support such a premise.

The absence of correlation between the age in years and cholesterol levels again was the expected result, the scatter of ages being narrow. Tanner (1951) reported positive correlation between the skinfold thickness and cholesterol concentrations, whilst Keys (1954) and

Keys and Fidanza (1960) observed such a correlation only in certain groups and other writers failed to detect such a correlation (Karvonen, Orma, Keys, Fidanza and Brozek, 1959; Thomas and Garn, 1960). The Malley Bay subjects showed no correlation between skinfold thickness and cholesterol levels or triglyceride concentrations, which is in agreement with the latter two groups quoted above. There was also no positive correlation between the maximum gain in body weight in a year and cholesterol and triglyceride levels; the maximum weight gain in young men over a length of time could act as an index of the proneness to obesity.

The significant correlation between serum total cholesterol, and beta-cholesterol with the cholesterol:phospholipid ratio is in agreement with the observations made at the end of the first part of Section VI. The significant correlation of cholesterol with total lipids is somewhat surprising in view of the degree of fluctuation shown by the latter. The correlation between total cholesterol and the cholesterol:phospholipid ratio was first noted by Peters and Man (1943).

From the description of the results it is clear that some of the subjects had more severe abnormalities in their serum than others. For example, subject RL showed evidence of raised triglycerides, raised total cholesterol, and an elevated proportion of cholesterol in the beta-lipoprotein fraction, and a raised cholesterol:phospholipid ratio. These findings betoken that the subject showed an aberrent mechanism for the transport of fats in the beta-lipoprotein fraction and also a possible faulty clearance of the chylomicron fraction. The subject consumed 45% of his dietary intake as fat; it seems, therefore, that in a man of 22 years with the above findings which were quite marked, preventive measures against the development of atheromatosis and

its complications should surely be employed.

Subjects DA and GB showed raised serum triglycerides, combined with evidence of abnormal levels of beta-lipoprotein cholesterol, though the total cholesterol was not raised. It may be that these subjects might have represented an earlier stage in the development of an aberrent lipid transport mechanism.

Subjects MJ, MTH and SM had raised triglyceride levels, with a raised cholesterol:phospholipid ratio. There was no elevation of the beta-cholesterol. It seems likely that these subjects were similar to subjects CD and BP and that the elevated cholesterol:phospholipid ratio should be ignored as meaningless, and the high levels of triglyceride should be regarded as secondary to the abnormally slow clearance of the fraction, probably in the chylomicron class.

Two subjects demonstrated an elevated total cholesterol concentration (DE and AP) but the lipoproteins appeared satisfactory. Except for the family history of ischaemic heart disease in the case of DE, it is unlikely that the elevated total cholesterol has predictive significance without additional evidence of abnormal mechanism in the serum lipoproteins.

In the remaining subjects (MBR, JS, ED and MJ) the abnormality occurred in the percentage of cholesterol in the beta-lipoprotein fraction, which was elevated. These may be early signs of more severe aberrations in the transport mechanism which might occur in later years.

In a report of the Framlingham study Kagan, Dawber, Kannel and Revotskie (1962) assessed the risk of contracting ischaemic heart disease expressed as morbidity ratios, i.e. the ratio of the observed number of cases to the expected number multiplied by 100. They showed that

increasing levels of serum cholesterol were found to be associated with increasing risk of developing coronary heart disease. Among males this gradient of risk held for those below and above the age of 50 years at entry, but the gradient was stronger in the younger group. Thus the risk of developing coronary heart disease in 8 years according to the initial cholesterol level for concentration above 260 mg % was shown by a morbidity ratio of 220 (twice the number which were expected) and for levels between 240 and 259 mg % it was shown by a ratio of 171. For subject RL at the age of 22 years it seems likely that the risk would be considerably greater, and similarly, but to a lesser extent, for subject DE and AP.

The thirteen subjects with some abnormality in their serial lipograms were compared as a group, with those subjects with no abnormality in the serum. The mean total calorie intake for the abnormal group was 3822 kcal/man/day and that for the remaining nine subjects was 3316 kcal/man/day. When these figures were computed per kilogram of body weight, they amounted to 47.4 and 47.6 kcal/kg respectively, and thus when the calorie intakes were standardised for body weight, the difference is seen to have been negligible. No differences were found in the maximum gain in body weight between the two groups, neither were there differences in the percentage intake of fat, or in systolic and diastolic blood pressure.

It is concluded, therefore, that though there are signs of aberrent lipid transport mechanism in the serum of a large proportion of the Halley Bay group, no group differences in the food intakes and fat intakes could be elucidated. That the dietary intake does influence the blood lipid picture has already been stressed; it is interesting that in the subjects with abnormality in the serum lipids, the dietary intake shows no difference from that of the group of normals.

One aim of this section is the attempt to discern how the abnormalities in the serum lipograms relate to the other variables measured. The only measurement which had any clinical significance, with the exception of the serum lipids, were the estimations of blood pressure and pulse rates, and not one of the latter could be said to have indicated abnormality. However, it was shown in the examination of the high and low triglyceride groups, that the blood pressure and pulse rates were slightly elevated in those subjects with high triglycerides.

The research worker is continually faced with the problem of defining the real boundaries of normality of the parameters he is measuring. In a parameter that can progessively change for the worse, is it possible to pick up such a change at an early stage? Clearly, it is impossible to say in terms of serum lipids with the facts that are known today. A way out of such a difficulty would be to make serial measurements of a quantity of parameters, all relating to the same disease, and, should slight derangements be manifest at an early stage which gain in magnitude as a function of advancing time, then the prognostic significance of those parameters has increased in value. The relationship between serum lipids and atheroma and ischaemic heart disease can only be finally proven by long term experiments which last several decades. However, the point remains that atheroma does occur in young men, as was shown in the autopsy studies made on casualties from the Korean War (Enos, Holmes and Beyer, 1953).

The present day approach to cardiovascular heart disease is based on the treatment of the already contracted illness, or the eradication of the disease from groups which have been shown to be highly affected. A third approach, and it is mainly a public health

one, should aim to sift out from an apparently normal population by biochemical and other estimations one which would appear to have a greater susceptibility to atheromatosis, and that group should be treated prophylactically by dietary methods and any other suitable means available.

Some of the subjects used in the present survey appeared to have well marked abnormalities in their lipograms. That the high incidence of aberrent mechanism in the serum lipids was due to conditions in Antarctica cannot be definitely stated; it has been already concluded that the percentage of calories supplied by fat did not greatly differ from estimations of the intake in the United Kingdom. It appears likely that the levels shown to exist in the group as a whole were a reasonable indication of the levels which would exist in any population of males of the same age range in the more civilised western countries. However, it is important to realise that it was the serial sampling over a period of 12 months which brought much of the abnormality to light. The interesting fact of the survey is that these abnormalities do occur in young people, and if long term studies show that the lipid levels are of significance in determining the future development of atheromatosis and ischaemic heart disease, then it is surely a powerful public health weapon.

It can also be envisaged from the opposite viewpoint that the occurrence of these raised lipid levels in the serum of young men is a strong argument against their having a share in the aetiology of ischaemic heart disease. This is particularly true when it is remembered that thirteen out of the twentyfour subjects taking part in the survey exhibited some form of lipid aberration.

3. Conclusions

i. The survey is disappointing due to the fact that though seasonal changes in the environmental outside temperatures caused well marked changes in the food intake and energy expenditure, the examination of the monthly means for seasonal effect was by and large negative. It is apparent that the lipid regulatory device is such that, in spite of the seasonal dietary and physical activity changes, the serum levels can be kept constant within a fairly narrow margin.

ii. However, in the case of the alpha- and beta-lipoprotein cholesterol levels, there were some changes which clearly related to the seasonal variations in climate. During the period defined as winter, the alpha-cholesterol level was depressed on the summer levels. During the same period the beta-cholesterol expressed as a percentage of the total cholesterol was raised when compared to the summer levels. Though these changes were only of marginal significance, and were of small amplitude, they are of particular interest in reference to the alpha-levels. Alpha-lipoprotein has been shown to be remarkably resistant to variation under the influence of dietary manipulation. It appeared more likely that the seasonal changes in alpha- and beta-cholesterol were related to seasonal changes in physical activity rather than to changes in dietary intake.

iii. The changes in the serum alpha- and beta-cholesterol levels in serum taken after sledging journeys suggested a similar manifestation; after the increased physical activity the alpha-cholesterol level rose and there was a depression of the beta-cholesterol.

iv. The total cholesterol levels demonstrated a slight downward trend during the 12 months of the survey. This was due largely to the serum levels of the first year subjects, the second year group

demonstrating a fall of approximately one third of that shown by the first year subjects. No clear conclusions could be made as to the cause of the downward trend of the monthly mean cholesterol levels; the fact that it was the first year subjects who showed the greatest fall suggests that the change could be an index of acclimatization and resulted from an alteration in endocrine balance.

v. Examination of the serum triglyceride levels showed that there was no monthly trend but demonstrated the existence of two significantly differing groups of subjects; one group with a mean level and a standard deviation of 231.3 $\pm$ 92.9 mg %, and a second group for whom the mean triglyceride concentration was 91.2 $\pm$ 31.4 mg %. Thus 2% of the population showed evidence of raised triglyceride concentrations. It is pointed out that if serum triglyceride levels should prove to be a reliable prognostic tool for assessing the risk of future ischaemic heart disease, then the fact that young men show such marked differences in the serial triglyceride concentrations will surely prove useful in combatting the disease in large scale public health surveys. The group with the high triglyceride levels were heavier, they tended to gain body weight to a greater extent, and their blood pressures and pulse rates were higher than the group with low triglyceride concentrations. Though the group means of the cholesterol levels, the cholesterol:phospholipid ratios and the total calorie intakes appeared to be markedly different, no significance could be established.

vi. Further examination of the individual serum levels showed that some degree of abnormality was present in eight more of the subjects; an attempt was made to demonstrate whether the abnormality lay in the beta-lipoprotein, or in the very low density class of lipoproteins. There were no group differences between the group with lipid abnormalities the group of normals in and/the dietary intake, degree of obesity and blood pressure, etc.

vii. There was a positive correlation between the percentage of calories supplied by fat and a) serum phospholipids b) serum cholesterol and beta-cholesterol, the latter expressed as absolute values and as a percentage of the total cholesterol, and c) the cholesterol: phospholipid ratio. Similarly positive correlation was found to exist between a) the total and the beta-cholesterol concentrations, and b) the cholesterol-phospholipid ratio and the absolute levels of dietary fat intake in calories. It is concluded that the habitual dietary intake of fat does have an influence on the personal serum lipid level.

SUMMARY

A survey was made on twentyfive young men at Halley Bay, situated on the coast of the Weddel Sea, Antarctica, over a period of one year. It involved serial observations at regular intervals on the following parameters:

1) the fatty acid composition of the subcutaneous adipose tissue
2) body weight, skinfold thickness and dietary intake
3) blood pressure and pulse rate
4) serum lipid concentration.

The year at Halley Bay is characterised by the occurrence of three well demarcated seasons, namely: 1) February (arrival) to April, when there is daylight, and temperatures range from -12.4 to -21.9°C; 2) May to September, when there is polar night, and outside temperatures vary between -23.3 and -34.9°C, and during this winter period the outside exposure and the energy expenditure is greatly reduced on summer levels; 3) October to January (departure) when the daylight returns and the outside temperatures range from -6.0 to -12.0°C, and the energy expenditure becomes increased. The object of the survey was to determine the influence of the seasonal changes in climate and activity on the various parameters and also to ascertain any significant groupings of the individual results.

The study of the composition of the subcutaneous fat indicated that:

1. On comparison with the compositions reported for other communities living in temperate regions, the differences were small and clearly non-significant. However, there remained the one marked dissimilarity in that the linolenic acid content of the adipose fat of the Halley Bay subjects composed an average of 2% of the total fatty

acids, whereas it had not been reported in significant amounts by other workers.

2. The intra-individual variations shown by each subject was surprisingly high, from which it might be construed that the adipose fat is by no means an inert energy store.

3. Variance analysis of oleic acid and the total unsaturated fatty acids revealed a seasonal effect to be significant at the 5% level. An increase of 1.8% in the amount of unsaturated fatty acids occurred between the first and the second seasons. It is possible that this may represent an index of acclimatization to cold in the human. Whether it merely accompanied the changes in body weight and skinfold thickness (vide infra) could not be stated.

From the data collected for the second part of the study, the following conclusions were drawn:

1. That the mean calorie intake was 3597 kcal/man/day, and that the percentage composition did not show any marked difference from the national average intake in the United Kingdom.

2. That there was a fall in total calories which occurred between the first summer and the winter, of 486 kcal ($p < 0.001$) which was accompanied by decrements in protein ($p < 0.001$), fat ($p < 0.01$) and carbohydrate calories ($p < 0.01$). A rise of 299 kcal occurring in total consumption between the second and the third season ($p < 0.05$) was emulated in the fat intake ($p < 0.01$) and to a lesser extent in the carbohydrate calories (NS) while there was a negligible rise in protein calories. A difference of 187 kcal between the food consumption of the first and second summers was non-significant, which was also the case for fat and carbohydrate calories. The difference between the

protein intakes for the two summer seasons was significant ($p < 0.001$).

The 'between men' effects in the statistical analysis of the four parameters was significant for total calories and for fat and carbohydrate calories ($p < 0.001$), but was non-significant for protein intake.

3. The calorie consumption of four occupational groups, namely manual workers, technicians, cooks and scientists was 4115, 3152, 3227 and 3729 kcal/man/day respectively. The only significant difference was between the manual workers and the technicians ($p < 0.02$) .

4. That the mean monthly body weight showed the maximum increase of 2.5 kg during the first 2 months of the survey (February to April), after which they levelled off and remained comparatively constant for the rest of the year. There was a very slight fall in the body weight in the last 4 months. From comparisons made between the dietary intake and body weight data, there is strong evidence to suggest that the gain in weight in the first 2 months of the year was due to a build up of protein or carbohydrate as well as the storage of adipose fat.

5. That the skinfold thickness readings at the four sites, namely lateral side of the arm, scapula, pectoral, and abdominal, showed their maximum increase in the first 3 months of the survey (February to May) and thus showed a dissociation from the period of maximum increase in body weight ($p < 0.001$). The anomalous result could not adequately be explained.

The results of the third part of the survey, the serial survey of blood pressure and pulse rates indicated a slight overall tendency for the systolic blood pressure to rise; however, there was superimposed on this rise a winter depression. The pulse pressure showed a tendency

to fall in the winter and to rise in the summer. The diastolic blood pressure and the pulse rate showed minimal changes.

The fourth part of the survey, which concerned the seasonal changes in the serum lipids, was in the main characterised by the absence of seasonal effects which might have been expected from work previously carried out by other workers.

The most important findings were:-

1. The average serum lipid levels for the whole year were: total lipids 655.6 $\pm$ 122.0 mg %; phospholipids 221.4 $\pm$ 20.7 mg %; triglycerides 120.8 $\pm$ 75.8 mg %. No seasonal trends were evident. There were marked subject differences in the level of serum triglycerides. Five of the subjects had a mean level of 231.3 $\pm$ 92.9 mg %, and nineteen had a mean of 91.2 $\pm$ 31.4 mg %.

2. Serum total cholesterol (199.0 $\pm$ 31.7 mg %) and beta-cholesterol (165.0 $\pm$ 33.4 mg %) were higher in the first half of the year than in the second half.

3. During the winter, when physical activity was at a minimum level the alpha-cholesterol was depressed, and the beta-cholesterol when expressed as a percentage of the total cholesterol was raised.

4. During sledging journeys there was a high fat intake, a high level of energy expenditure, and some weight loss. Fasting samples taken on return to base showed an elevation of alpha-cholesterol, and a depression of beta-cholesterol levels. The occurrence of these changes during the active portions of the year and following sledging journeys suggested that they were more dependent upon levels of energy expenditure than on alterations of the dietary intake.

5. In a detailed examination of the lipid concentrations of each individual it was concluded that thirteen out of the twentyfour subjects showed some abnormality. No inter-group difference could be found in the composition of the dietary intake, the amount of obesity, the maximum amount of weight gained during the year and the blood pressure and pulse rate.

6. There was a positive correlation between the total calorie fat intake and the percentage of the total consumption furnished by fat and the following lipid parameters: a) total cholesterol levels b) beta-cholesterol levels c) the cholesterol: phospholipid ratios.

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* Read in abstract form.

APPENDIX TO SECTION III

Table 38 . The fatty acid compositions of adipose biopsies taken from 19 subjects, in which the fatty acids have been expressed as a percentage of the total. The fatty acids C12:0, C12:1, and C17:0 have been omitted from the results.

Subject DA

Month	C14:0	C14:1	C16:0	C16:1	C18:0	C18:1	C18:2	C18:3
February 1961	3.9	1.5	24.4	10.0	4.6	42.2	4.9	3.2
May	2.4	1.5	22.3	7.6	5.3	51.5	6.1	2.6
June	5.0	1.9	21.9	11.3	3.2	45.6	6.5	2.0
July	3.9	1.6	20.8	10.3	2.7	50.3	6.4	2.2
August	4.2	1.6	22.7	9.4	3.3	50.2	5.0	2.0
October	2.8	0.9	22.4	7.6	4.1	53.8	5.6	2.4
November	3.9	1.3	26.6	8.5	4.6	46.7	5.0	2.5
January 1962	4.9	2.0	23.5	9.0	4.1	43.9	4.1	2.7
Subject GB								
March	2.6	1.9	20.7	11.5	4.2	47.1	9.6	1.6
April	3.0	3.0	23.9	10.3	6.0	42.9	9.2	-
May	3.1	1.5	20.9	12.0	3.7	44.2	8.2	2.0
June	1.4	1.3	19.5	11.6	2.5	53.9	8.6	1.6
July	4.5	1.6	20.8	11.1	3.6	47.4	8.8	1.9
January 1962	2.6	tr	21.9	11.0	3.7	45.7	8.7	3.3
Subject MBR								
March	3.1	1.5	26.8	8.6	4.6	40.8	11.9	1.1
May	3.3	0.8	24.1	8.6	4.2	47.3	8.2	1.5
July	4.8	1.0	24.9	8.2	5.5	46.8	6.3	1.6
August	5.6	0.6	24.4	8.1	5.9	45.7	6.9	1.6
October	5.4	1.2	24.9	9.3	4.6	45.6	6.1	1.9
November	5.1	1.7	22.5	9.8	4.0	45.5	7.5	2.1
January 1962	4.7	1.2	24.7	8.9	4.6	45.5	6.3	1.9

Subject CD

Month	C14:0	C14:1	C16:0	C16:1	C18:0	C18:1	C18:2	C18:3
March	2.2	1.4	24.1	8.7	5.2	49.1	6.0	1.0
July	3.8	0.7	23.2	8.1	3.6	51.8	6.2	1.3
September	3.2	0.7	23.7	8.5	2.6	49.0	7.7	1.6
October	2.5	1.9	25.7	8.1	4.3	49.1	5.8	1.1
January 1962	2.9	1.3	21.8	7.8	4.6	52.6	5.9	1.9
Subject ED								
February	4.1	1.0	21.7	7.8	4.7	48.5	9.2	1.9
May	3.6	1.6	23.8	10.4	2.3	49.9	4.7	1.7
July	1.9	1.9	23.7	6.6	5.1	53.8	6.8	tr
September	3.3	2.89	22.7	7.0	3.6	48.8	7.3	2.0
December	4.2	2.1	12.8	2.0	6.2	56.7	12.0	3.1
Subject DE								
February	4.2	1.2	26.2	6.1	4.6	48.7	5.8	1.6
August	1.9	1.3	21.9	10.7	3.3	49.4	8.2	3.3
October	2.9	0.7	23.1	9.9	4.1	49.6	6.7	2.0
November	3.0	3.9	19.6	7.1	5.3	42.3	5.6	3.9
December	2.2	1.0	23.9	6.8	7.2	48.7	7.5	1.7
January	1.4	1.0	22.3	6.7	4.1	55.5	7.1	1.4
Subject MJ								
February	5.4	1.6	25.6	13.6	5.4	38.4	6.9	2.7
April	4.0	1.7	23.2	12.1	4.1	46.3	5.9	2.2
May	4.0	1.7	21.2	11.8	2.8	49.9	6.2	1.8
June	3.2	1.6	19.4	10.4	2.6	55.1	5.4	1.8
July	3.4	1.6	21.7	11.3	3.6	48.6	6.2	2.5
September	3.1	1.5	22.4	9.1	3.4	51.1	6.1	1.9
October	2.7	1.6	21.0	9.7	4.3	51.2	6.5	2.8

Subject DJ

Month	C14:0	C14:1	C16:0	C16:1	C18:0	C18:1	C18:2	C18:3
March	3.1	1.5	22.7	9.5	4.3	49.9	5.9	2.9
May	4.4	1.7	24.5	10.8	4.3	45.7	5.7	2.4
June	5.0	2.9	26.0	10.2	5.0	43.1	5.0	1.9
August	2.7	0.7	21.9	11.1	4.4	46.9	6.2	5.1
September	3.9	1.2	23.3	9.7	4.9	47.8	5.6	2.3
December	4.6	1.8	23.4	9.8	4.5	47.8	5.5	1.9
January 1962	2.9	1.8	25.7	9.0	4.0	43.1	6.5	2.1
Subject RL								
February	4.6	2.1	20.1	8.9	4.9	45.6	8.5	2.4
March	2.1	0.7	21.0	8.1	3.4	55.3	7.3	1.8
May	1.0	0.5	16.0	6.8	4.4	61.0	7.1	2.9
June	2.4	1.0	17.6	7.1	4.2	57.8	7.4	2.3
July	3.0	1.1	21.8	8.0	5.7	52.0	6.6	1.9
August	2.7	1.6	20.4	8.0	4.6	53.2	6.8	2.4
September	2.6	1.1	19.7	7.5	4.5	54.6	7.1	2.6
October	2.5	4.3	20.0	8.5	4.4	43.1	6.6	6.4
November	2.4	1.1	21.9	7.0	6.2	49.7	6.4	4.1
December	2.1	tr	21.9	5.9	5.9	51.7	6.9	5.7
January1962	3.8	1.3	25.1	6.6	5.0	49.6	5.4	2.1
Subject SM								
February	1.5	3.8	16.9	5.6	5.9	44.3	8.2	3.1
March	0.3	0.5	15.1	7.7	5.1	58.6	9.3	3.5
April	0.6	2.3	18.4	7.4	7.6	49.0	8.6	3.9
June	2.9	3.5	18.1	7.6	4.1	47.3	8.3	2.7
July	2.8	1.5	22.2	9.2	3.9	50.6	6.4	3.0
August	3.9	5.3	20.3	10.9	5.5	42.1	6.4	2.7
October	2.5	1.3	19.6	9.2	3.8	53.8	6.4	2.9
November	2.2	2.1	21.4	4.9	4.7	49.9	6.7	6.2
December	3.1	2.2	24.4	7.5	4.2	47.5	5.5	2.5
January	3.5	2.0	24.5	5.9	5.7	40.7	4.8	4.1

Subject CM

Month	C14:0	C14:1	C16:0	C16:1	C18:0	C18:1	C18:2	C18:3
February	4.0	2.1	22.1	4.9	4.0	51.9	7.4	1.7
April	2.2	1.4	24.5	11.1	5.3	45.7	5.0	2.5
May	3.5	1.0	23.2	10.0	2.8	51.4	6.0	1.2
July	3.9	1.2	24.5	10.1	1.5	51.1	5.0	1.9
August	3.2	1.1	22.0	9.4	2.6	51.8	6.2	2.8
September	2.0	0.8	20.6	8.3	4.0	56.2	5.6	2.0
November	4.6	3.1	23.6	10.7	2.5	47.8	5.8	1.2
November	3.9	1.5	21.0	10.3	2.8	51.1	6.4	1.9
December	3.7	1.0	24.0	6.8	3.5	50.7	6.7	2.0
January 1962	4.2	3.2	25.0	8.6	3.3	48.5	5.7	0.8
Subject PM								
February	6.7	3.9	24.4	8.7	4.2	42.2	5.5	1.9
April	4.5	3.6	21.6	8.6	4.0	48.1	5.2	1.3
May	2.8	1.1	22.0	8.3	2.9	55.6	5.3	1.3
July	3.4	1.1	22.8	9.2	3.1	50.4	6.8	1.8
September	5.5	2.1	21.8	7.9	3.8	45.4	9.1	1.3
January 1962	3.7	1.5	22.8	9.1	2.5	51.5	5.5	1.5
Subject AP								
February	3.5	1.3	25.5	8.9	6.8	46.0	6.2	1.8
April	2.5	0.4	21.5	10.4	6.3	50.1	6.4	2.1
May	3.2	1.7	20.6	8.6	7.8	50.8	5.0	2.2
June	5.9	1.6	22.4	12.7	7.2	46.0	5.7	1.3
July	3.0	1.5	20.5	11.0	4.2	51.5	6.5	1.5
August	3.0	1.4	27.4	8.9	6.4	50.0	5.0	1.2
September	3.4	tr	25.8	9.3	4.4	50.4	5.3	0.6
October	3.6	1.1	23.2	9.4	4.3	51.3	6.4	tr
January 1962	2.5	1.3	18.2	7.5	7.0	56.7	5.4	1.4

Subject [illegible]	C14:0	C14:1	C16:0	C16:1	C18:0	C18:1	C18:2	C18:3
February	3.6	1.2	21.4	7.6	6.2	48.5	9.1	2.4
April	3.1	0.8	21.1	7.1	5.2	54.2	5.6	1.7
May	4.0	tr	23.9	9.2	4.7	50.4	5.9	2.1
July	3.1	0.1	26.3	11.7	4.1	41.7	7.3	3.3
September	4.2	1.1	27.5	7.6	5.9	44.7	6.8	1.5
October	3.4	0.8	22.4	9.2	4.9	49.3	6.3	3.2
January 1962	4.0	1.4	22.4	10.2	3.7	48.8	6.6	1.9
Subject JS								
February	4.8	1.5	23.8	8.9	5.2	43.9	7.9	1.5
April	4.5	0.9	24.2	8.4	4.4	48.6	5.3	1.7
July	6.1	1.7	23.2	10.3	4.1	43.2	7.0	1.8
September	3.1	1.3	22.3	10.3	4.3	48.2	7.0	2.9
October	2.8	1.1	22.9	10.1	4.2	51.2	4.5	2.0
December	0.8	0.4	19.1	6.3	5.9	58.0	6.8	2.5
January 1962	4.5	1.0	23.7	8.4	4.3	49.9	4.7	2.0
Subject ES								
February	4.4	0.9	23.2	11.5	4.3	45.7	6.4	2.6
April	4.9	1.7	24.5	10.1	5.1	44.0	5.1	2.6
June	3.7	1.6	22.8	9.7	3.6	47.9	6.1	2.8
August	4.0	0.7	22.3	10.4	4.9	47.5	6.0	2.8
September	5.2	2.1	22.5	12.2	3.4	42.3	7.3	2.9
November	4.6	1.1	24.9	7.5	5.1	48.8	5.1	1.8
January 1962	4.0	0.8	23.5	9.5	3.7	49.0	5.7	2.8

Subject GT

Month	C14:0	C14:1	C16:0	C16:1	C18:0	C18:1	C18:2	C18:3
February	1.1	0.8	24.5	4.1	3.8	62.6	2.3	0.8
April	3.6	2.2	24.1	5.1	6.0	54.0	4.0	tr
June	1.5	1.5	23.8	4.7	5.4	58.6	4.5	tr
August	2.3	1.8	30.0	6.0	7.9	46.3	5.8	tr
September	2.7	0.4	22.2	6.4	4.1	57.1	5.1	0.7
October	1.1	0.2	21.7	4.8	5.1	60.9	6.2	tr
Subject ET								
March	3.4	0.9	23.8	8.3	2.6	52.1	5.9	2.2
April	3.2	2.1	21.7	11.1	3.7	48.9	5.7	1.8
July	3.4	-	23.1	8.7	5.6	49.8	4.6	2.2
August	5.3	1.3	21.4	12.1	4.7	42.6	8.7	2.2
September	2.8	1.2	22.7	9.9	3.4	52.8	4.1	2.0
October	1.4	0.5	19.6	7.9	3.9	56.6	7.5	2.1
November	1.8	0.9	19.8	7.8	6.0	55.8	4.6	2.3
January 1962	3.2	1.1	22.8	8.6	3.1	51.7	5.6	2.9
Subject MTH								
February	5.2	2.2	23.2	12.2	3.3	45.0	3.9	2.5
April	3.6	0.7	27.0	11.5	4.5	43.9	4.8	2.2
June	3.5	1.1	22.5	9.2	4.4	48.7	6.6	2.7
July	4.2	0.9	23.8	6.9	4.2	49.8	7.3	2.1
September	3.1	0.9	19.9	9.4	4.0	52.5	7.0	1.9
November	4.2	0.9	25.8	8.4	3.4	46.3	6.9	1.6
December	2.8	0.7	24.9	7.4	6.8	44.5	7.0	1.8

Table 39. The mean individual fatty acid composition of subcutaneous fat biopsies taken in the first season, February to April.

Subject	Fatty Acid Short Term Denomination							
	14:0	14:1	16:0	16:1	18:0	18:1	18:2	18:3
MTH	4.4	1.4	25.1	11.8	3.9	44.5	4.3	2.3
ED	4.1	1.0	21.7	7.8	4.7	48.5	9.2	1.9
MBR	3.1	1.5	26.8	8.6	4.6	40.8	11.9	1.1
DJ	3.1	1.5	22.7	9.5	4.3	49.9	5.9	2.9
PN	5.6	3.7	23.0	8.6	4.1	45.1	5.3	1.6
SM	0.8	2.2	16.8	6.9	6.2	50.6	8.7	3.5
GB	2.8	2.4	22.3	10.9	5.1	45.0	9.4	1.6
RL	3.3	1.4	20.5	8.5	4.1	50.2	7.9	2.1
DA	3.9	1.5	24.4	10.0	4.6	42.2	4.9	3.2
AP	3.0	0.8	23.5	9.7	6.6	48.1	6.3	1.9
JS	4.6	1.1	24.0	8.6	4.8	46.2	6.6	1.6
CD	2.2	1.4	24.1	8.7	5.2	49.1	6.0	1.0
MS	4.7	1.3	23.8	10.8	4.7	44.9	5.8	2.6
GM	3.1	1.2	23.3	8.0	4.6	48.8	6.2	2.1
ET	3.3	1.5	22.7	9.7	3.1	50.5	5.8	2.0
BP	3.4	1.0	21.7	7.3	5.7	51.3	7.3	2.0
MJ	4.7	1.6	24.4	12.8	4.7	42.3	6.4	2.4
GT	2.3	1.5	24.3	4.6	4.9	58.3	3.1	-
DE	4.2	1.2	26.2	6.1	4.6	48.7	5.8	1.6

Table 40. The mean individual percentage composition of fat biopsies taken from the subcutis, for the second season, May to September.

Subject	Fatty Acid Short Term Denomination							
	14:0	14:1	16:0	16:1	18:0	18:1	18:2	18:3
MTH	3.6	0.9	22.0	8.5	4.2	50.3	6.9	2.2
ED	2.9	2.1	23.4	8.0	3.7	50.8	6.2	1.3
MBR	4.5	0.8	24.4	8.3	5.2	46.6	7.1	1.5
DJ	4.0	1.6	23.9	10.5	4.7	45.8	5.6	2.9
PN	3.9	1.4	22.2	8.4	3.3	50.5	7.0	1.4
SM	3.2	3.4	20.2	9.2	4.5	46.6	7.0	2.8
GB	3.0	1.4	20.4	11.5	3.3	48.5	8.5	1.8
RL	2.3	1.0	19.1	7.5	4.7	55.7	7.0	2.4
DA	3.9	1.6	21.9	9.7	3.6	49.4	6.0	2.2
AP	3.7	1.6	23.3	10.1	6.0	47.7	5.5	1.4
JS	4.6	1.5	22.8	10.3	4.2	45.7	7.0	2.3
CD	3.2	1.1	24.3	8.2	3.5	49.9	6.6	1.3
MS	4.3	1.4	22.8	10.7	3.9	45.9	6.5	2.8
GM	3.1	1.0	22.5	9.4	2.7	52.1	5.7	2.0
ET	3.8	1.2	22.4	10.3	4.9	48.4	5.8	2.2
BP	3.8	0.6	25.9	9.5	4.9	45.6	6.7	2.3
MJ	3.4	1.5	21.2	10.7	3.1	51.2	5.8	2.0
GT	2.2	1.2	25.3	5.4	5.5	54.0	5.1	-
DE	1.9	1.3	21.9	10.7	3.3	49.4	8.2	3.3

Table 41. The mean individual percentage composition of adipose fat biopsies for the third season, October to January.

Subject	Fatty Acid Short Term Denomination							
	14:0	14:1	16:0	16:1	18:0	18:1	18:2	18:3
MTH	3.5	0.8	25.3	7.9	5.1	45.4	7.0	1.7
ED	4.2	2.1	12.8	2.0	6.2	56.7	12.0	3.1
MBR	5.0	1.3	24.0	9.3	4.4	45.5	6.6	1.9
DJ	3.7	1.8	24.5	9.4	4.2	45.4	6.0	2.0
PN	3.7	1.5	22.8	9.1	2.5	51.5	5.5	1.5
SM	2.8	1.9	22.9	6.9	4.6	47.9	5.8	3.9
GB	2.6	TR	21.9	11.0	3.7	45.7	8.7	3.3
RL	2.7	2.2	22.2	7.0	5.4	48.5	6.3	4.5
DA	3.8	1.4	24.2	8.4	4.3	48.1	4.9	2.5
AP	3.0	1.2	20.7	8.5	5.2	54.0	5.9	1.4
JS	2.7	0.8	21.9	8.3	4.8	53.0	5.3	2.2
CD	2.9	1.3	21.8	7.8	4.6	52.6	5.9	1.9
MS	4.3	0.9	24.2	8.5	4.4	48.9	5.4	2.3
GM	4.1	2.2	23.4	9.1	3.0	49.5	6.2	1.5
ET	2.1	0.8	20.7	8.1	4.3	54.7	5.9	2.4
BP	3.7	1.1	22.4	9.7	4.3	49.1	6.4	2.5
MJ	2.7	1.6	21.0	9.7	4.3	51.2	6.5	2.8
GT	1.1	0.2	21.7	4.8	5.1	60.9	6.2	-
DE	2.4	1.7	22.2	7.6	5.2	49.0	6.7	2.3

Table 42 - 44. The results of the survey of adipose fat changes treated by analysis of variance, to determine the significance of the seasonal effects suggested from the meaned values.

Table 42 . Oleic Acid (C 18 : 1)

Source of Variation	Sum of squares	Degrees of freedom	Mean square	Variance ratio	Significance
Between Men	455.74	18	25.319	3.000	0.5 %
Between Season	73.08	2	36.584	4.32	5 %
Residual	304.27	36	8.452	-	-
Total	833.09	56	-	-	-

Table 43. Total unsaturated fatty acids.

Source of Variation	Sum of squares	Degrees of freedom	Mean square	Variance ratio	Significance
Between men	521.4	18	28.9	5.57	0.1 %
Between seasons	37.4	2	18.7	3.596	5 %
Residual	187.2	36	5.20	-	-
Total	746.0	56	-	-	-

Table 44. Total saturated fatty acids.

Source of variation	Sum of squares	Degrees of Freedom	Mean square	Variance ratio	Significance
Between men	152.0	18	8.44	1.90	5 %
Between seasons	18.7	2	9.36	2.112	NS
Residual	159.8	36	4.43	-	-
Total	330.5	56	-	-	-

APPENDIX TO SECTION IV

Table 45. The individual readings of the body weight for 24 subjects at monthly intervals (kg)

Subject	Feb	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan
DJ	66.10	66.65	67.57	68.85	65.20	66.42	65.81	65.17	66.75	66.65	67.15	66.20
AM	66.25	68.90	69.15	68.67	68.45	67.61	67.95	68.45	67.90	67.45	67.65	68.80
ET	66.10	67.25	69.20	69.15	70.45	69.73	69.90	70.40	69.20	68.40	-	69.30
MS	61.85	63.55	63.37	63.45	63.80	63.45	64.30	64.35	64.20	63.65	62.50	64.35
GT	60.40	61.45	61.80	61.60	61.65	61.27	62.30	62.70	62.00	62.25	61.00	62.55
CJ	72.05	73.20	74.85	76.90	75.10	74.85	75.50	73.50	75.30	-	72.65	75.30
RL	95.40	97.10	97.50	97.97	97.85	97.00	97.05	98.00	96.70	99.15	98.80	101.25
JS	66.70	67.70	70.00	68.30	68.40	68.75	69.34	69.90	70.05	69.35	69.75	68.45
AP	68.20	70.85	69.95	71.40	72.20	73.00	73.43	72.65	72.50	72.35	71.60	69.50
DED	93.90	94.80	97.60	98.40	96.00	96.90	97.25	99.33	99.45	97.30	95.65	98.10
MJ	67.00	68.65	72.55	71.95	72.25	71.75	71.65	72.45	71.80	73.70	72.25	70.25
DE	73.10	71.40	75.45	-	77.30	77.45	76.15	75.70	75.50	75.87	75.55	74.20
DA	81.70	83.20	83.65	84.30	85.45	85.76	86.65	86.95	85.34	-	81.50	83.90
GB	100.35	100.15	100.75	101.75	101.40	101.85	102.45	101.95	102.80	103.75	103.10	101.60
MB	69.90	71.05	71.10	70.95	71.95	73.17	73.40	74.30	75.60	74.60	71.55	72.45
PN	72.40	76.10	77.67	77.45	77.35	78.77	78.75	77.45	77.75	78.45	78.50	77.80
BP	66.90	69.10	70.05	70.30	70.50	71.65	70.65	71.975	69.90	70.35	69.40	69.60
SM	62.00	63.35	64.15	65.10	62.10	62.64	63.50	64.85	65.02	64.12	62.90	64.50
GM	70.90	71.70	74.15	75.05	75.35	75.00	76.35	74.25	76.35	75.05	75.35	76.80
CD	87.15	86.95	88.77	88.475	89.40	89.35	91.30	91.30	90.10	90.55	91.15	92.80
ED	80.30	79.95	81.35	80.85	80.30	80.10	80.55	79.70	79.35	78.80	77.65	77.95
MBR	-	71.35	72.90	73.50	73.45	73.75	74.73	75.05	74.75	74.35	75.70	73.95
MT	77.60	75.24	79.37	80.30	81.40	82.05	82.83	80.20	79.90	80.65	82.30	80.55
MTH	85.40	85.55	88.55	-	86.90	86.55	85.82	87.20	-	88.50	87.60	88.40
EJ	77.8	-	-	80.65	-	81.7	82.73	81.25	-	81.85	81.05	80.30

Table 46. Body weights before, directly after and one month after sledging trips (in kg)

Subject	Trip	Before Trip	After Trip	1 month After Trip
DA	1	83.2	81.25	83.65
	2	84.25	79.80	84.30
	3	85.34	81.50	83.90
MT	1	87.60	85.50	88.40
DED	1	97.30	95.65	98.10
AP	1	70.85	69.15	69.95
	2	72.05	67.30	71.40
	3	71.60	69.50	-
MT	1	82.75	80.20	79.90
CJ	1	73.20	72.65	76.90
	2	74.275	73.50	
	3	75.30	72.65	75.30
MS	1	63.25	62.50	64.35
GT	1	62.55	62.00	62.25
ET	1	68.40	68.26	69.30
JS	1	69.75	68.15	68.45
ED	1	78.8	77.65	77.95
SM	1	62.90	64.50	-
BP	1	69.40	69.60	-
MB	1	74.60	71.55	72.45
DE	1	71.40	72.6	75.45
	2	76.30	73.15	75.55
	3	75.55	74.20	-
GM	1	77.03	72.36	75.35
	2	75.35	76.80	-

Tables 47 to 58. The skinfold thickness measurements as the log transform, for the five sites on the body surface.

Table 47. FEBRUARY

Name	Site I	II	III	IV	V
DA	193	202	200	192	198
MB	154	202	193	163	212
GB	-	-	-	-	-
MBR	159	201	196	191	212
CD	176	198	207	185	197
ED	170	196	186	189	181
DE	128	175	179	156	178
DED	143	196	193	177	158
MJ	123	168	183	152	179
DJ	163	186	181	177	189
CJ	136	151	180	168	165
EJ	176	164	200	185	183
RL	181	222	203	208	-
SM	118	165	178	143	175
AM	-	-	-	-	-
GM	161	181	193	198	203
PN	186	202	197	188	212
BP	132	176	178	154	186
AP	128	185	197	161	149
JS	169	184	192	176	188
MS	154	185	181	163	183
GT	132	181	180	151	178
MT	176	188	204	191	199
ET	-	-	-	-	-
MTH	170	174	195	176	185

Table 48

MARCH

Name	Site I	II	III	IV	V
DA	196	193	206	199	206
MB	166	187	194	168	202
GB	-	-	-	-	-
MBR	166	207	196	183	203
CD	190	198	215	190	201
ED	172	189	189	184	176
DE	151	190	181	179	182
DED	148	185	196	176	154
MJ	130	170	187	174	173
DJ	160	170	183	180	195
CJ	138	159	192	149	177
EJ	-	-	-	-	-
RL	174	223	208	211	-
SM	126	162	184	167	176
AM	-	-	-	-	-
GM	169	184	201	210	203
PN	185	215	200	201	220
BP	143	169	180	169	200
AP	134	183	197	171	156
JS	164	188	191	181	187
MS	158	179	183	169	186
GT	130	163	181	159	158
MT	171	187	209	196	183
ET	153	195	199	186	196
MTH	164	183	202	190	187

Table 49.

APRIL

Name	Site I	II	III	IV	V
DA	180	208	204	190	198
MB	156	173	195	171	221
GB	-	-	-	-	-
MBR	168	205	200	188	200
CD	179	205	213	193	198
ED	185	194	190	195	178
DE	156	207	177	169	181
DED	173	190	198	191	151
MJ	138	183	195	192	198
DJ	158	175	181	181	188
CJ	134	165	188	151	179
EJ	-	-	-	-	-
RL	178	215	208	223	-
SM	130	167	190	161	181
AM	-	-	-	-	-
GM	176	198	203	215	201
PN	195	214	203	214	218
BP	140	185	185	156	195
AP	141	172	196	162	138
JS	164	191	193	188	170
MS	151	176	183	166	170
GT	126	168	187	154	162
MT	177	189	203	203	190
ET	146	194	199	182	181
MTH	178	186	206	186	190

Table 50.

MAY

Name	Site I	II	III	IV	V
DA	192	212	210	200	203
MB	158	204	199	175	209
GB	-	209	236	246	203
MBR	178	213	208	191	208
CD	194	210	220	195	198
ED	183	196	191	191	179
DE	154	218	190	169	179
DED	165	193	205	197	164
MJ	146	181	196	188	203
DJ	162	176	187	181	181
CJ	146	177	193	154	189
EJ	179	208	209	187	201
RL	176	216	213	216	-
SM	132	176	188	161	191
AM	-	-	-	-	-
GM	189	195	205	222	209
PN	192	217	205	196	220
BP	141	181	186	169	203
AP	143	187	202	174	146
JS	171	186	191	189	168
MS	153	182	184	168	181
GT	138	167	187	157	157
MT	186	194	210	217	190
ET	149	202	202	190	191
MTH	-	-	-	-	-

Table 51.

JUNE

Name	Site I	II	III	IV	V
DA	200	218	212	206	202
MB	159	186	193	173	219
GB	-	206	234	245	197
MBR	191	196	200	185	205
CD	186	213	220	196	199
ED	179	197	190	181	176
DE	149	223	184	176	181
DED	162	192	202	190	167
MJ	138	185	194	178	205
DJ	153	167	185	165	163
CJ	145	176	193	154	175
EJ	-	-	-	-	-
RL	171	214	208	216	-
SM	118	159	183	153	187
AM	-	-	-	-	-
GM	194	181	206	223	205
PN	190	205	203	197	220
BP	130	186	185	166	198
AP	146	187	203	189	140
JS	167	176	190	177	164
MS	156	179	183	174	184
GT	134	167	192	162	174
MT	188	191	214	217	190
ET	152	201	204	192	196
MTH	169	196	205	188	190

Table 52.

JULY

Name	Site I	II	III	IV	V
DA	203	215	212	199	205
MB	176	192	199	173	218
GB	-	207	236	243	193
MBR	180	199	209	198	219
CD	200	204	222	203	200
ED	176	193	191	190	179
DE	146	221	186	171	164
DED	169	193	200	192	151
MJ	132	189	198	186	198
DJ	157	164	182	170	165
CJ	154	173	191	151	174
EJ	176	194	212	194	195
RL	176	219	209	215	-
SM	120	167	184	154	189
AM	145	179	185	165	146
GM	185	181	209	223	204
PN	198	210	204	203	225
BP	141	182	188	167	205
AP	148	188	203	176	136
JS	163	173	191	175	176
MS	160	175	185	188	184
GT	138	168	189	164	154
MT	193	194	215	221	194
ET	157	201	203	200	203
MTH	179	200	204	198	191

Table 53.

AUGUST

Name	Site I	II	III	IV	V
DA	206	219	211	184	201
MB	177	198	203	176	219
GB	-	208	232	254	170
MBR	189	210	204	187	211
CD	185	209	220	199	195
ED	179	197	190	181	177
DE	154	210	185	166	186
DED	167	196	202	183	151
MJ	136	176	193	182	192
DJ	149	179	183	169	167
CJ	157	172	190	157	176
EJ	176	206	208	194	192
RL	161	216	207	193	-
SM	123	175	186	158	193
AM	130	181	181	162	165
GM	186	210	207	226	200
PN	195	216	204	198	222
BP	143	184	188	170	197
AP	154	196	204	192	140
JS	169	176	193	176	164
MS	160	157	185	176	183
GT	146	172	188	162	154
MT	190	194	213	215	190
ET	162	205	200	196	214
MTH	176	201	202	190	190

Table 54.

SEPTEMBER

Name	Site I	II	III	IV	V
DA	208	219	211	199	200
MB	182	203	206	175	222
GB	-	204	233	255	190
MBR	193	208	206	189	214
CD	195	209	220	202	195
ED	179	195	189	183	176
DE	149	219	187	175	186
DED	169	198	204	191	148
MJ	141	174	198	189	195
DJ	158	171	185	177	162
CJ	154	176	192	153	174
EJ	173	204	206	203	192
RL	167	219	209	208	-
SM	128	175	186	164	191
AM	141	177	183	170	148
GM	185	186	206	222	199
PN	195	216	204	198	222
BP	141	185	185	164	195
AP	153	188	203	182	132
JS	166	184	194	176	169
MS	161	186	185	166	185
GT	143	174	191	163	154
MT	197	202	221	212	189
ET	161	205	200	196	214
MTH	176	203	205	192	189

Table 55.

OCTOBER

Name	Site I	II	III	IV	V
DA	205	221	213	196	201
MB	183	199	206	176	227
GB	-	203	231	254	191
MBR	184	206	203	181	209
CD	198	210	218	203	194
ED	182	189	188	186	176
DE	151	209	182	175	178
DED	167	196	202	183	151
MJ	140	168	194	181	190
DJ	158	174	184	176	165
CJ	145	174	190	149	176
EJ	-	-	-	-	-
RL	171	217	204	210	-
SM	120	176	189	159	199
AM	138	176	185	163	145
GM	190	191	208	221	197
PN	200	214	206	193	227
BP	138	183	185	160	195
AP	154	194	203	183	130
JS	165	187	194	176	159
MS	159	182	186	183	181
GT	136	176	192	163	163
MT	179	189	212	209	183
ET	159	198	200	195	197
MTH	-	-	-	-	-

Table 56.

NOVEMBER

Name	Site I	II	III	IV	V
DA	206	214	210	197	203
MB	183	202	208	182	224
GB	-	208	237	249	192
MBR	196	213	208	187	217
CD	201	210	218	199	198
ED	180	189	189	185	183
DE	153	190	184	162	177
ÐED	167	196	201	187	189
MJ	141	177	198	186	190
DJ	166	179	183	177	173
CJ	-	-	-	-	-
EJ	172	207	210	193	197
RL	179	220	209	218	-
SM	138	180	187	164	193
AM	138	178	181	158	160
GM	184	186	206	218	199
PN	197	214	207	196	227
BP	141	185	187	162	191
AP	157	194	206	182	134
JS	169	185	193	180	183
MS	166	185	189	186	189
GT	146	176	191	168	172
MT	200	199	209	210	186
ET	157	194	201	199	203
MTH	173	201	206	185	188

Table 57.

DECEMBER

Name	Site I	II	III	IV	V
DA	198	212	206	194	202
MB	178	200	200	172	229
GB	-	201	237	240	192
MBR	192	210	207	183	217
CD	192	209	212	205	191
ED	179	193	187	189	180
DE	-	-	-	-	-
DED	167	190	200	183	154
MJ	140	174	195	186	195
DJ	159	179	185	176	174
CJ	148	170	188	162	182
EJ	166	199	204	202	200
RL	170	221	204	223	-
SM	123	176	184	160	191
AM	138	173	182	162	163
GM	186	201	207	220	195
PN	205	219	208	198	222
BP	134	180	185	161	197
AP	159	198	204	178	138
JS	163	188	193	179	183
MS	156	176	183	178	179
GT	141	176	194	176	176
MT	180	198	210	216	198
ET	153	199	198	188	205
MTH	179	194	202	182	186

Table 58 .

JANUARY

Name	Site I	II	III	IV	V
DA	200	210	208	198	200
MB	173	200	200	173	219
GB	-	203	237	240	189
MBR	191	211	207	188	213
CD	203	206	222	201	202
ED	180	191	187	188	181
DE	136	186	180	167	166
DED	168	197	203	193	160
MJ	138	172	191	180	196
DJ	170	178	183	172	172
CJ	158	178	193	163	178
EJ	165	199	205	186	196
RL	179	225	205	223	-
SM	120	167	183	154	191
AM	145	179	184	151	158
GM	192	186	209	218	196
PN	200	218	205	202	225
BP	136	178	183	157	199
AP	148	191	200	176	145
JS	166	184	190	176	173
MS	163	183	185	179	183
GT	146	176	189	172	170
MT	194	196	206	215	187
ET	161	199	200	186	203
MTH	177	194	204	179	187

Table 59. The mean skinfold thickness measurements as the log transform , with standard deviations.

Name	Site I		Site II		Site III		Site IV	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
DA	198.9	7.6	211.7	8.0	208.6	3.8	196.2	5.4
MB	170.4	10.7	195.5	8.9	199.7	5.0	173.1	4.4
GB	-	-	205.4	2.6	234.7	2.2	247.3	5.6
MBR	182.2	11.7	206.6	5.3	203.7	4.5	187.6	4.4
CD	191.6	8.3	206.7	4.6	217.3	4.4	197.6	5.8
ED	178.7	4.1	193.3	3.0	189.0	1.5	186.8	4.1
DE	147.9	8.1	204.4	15.7	183.1	3.6	169.5	6.4
DED	163.7	8.6	193.5	3.6	200.5	3.3	186.1	6.1
MJ	136.9	5.8	176.4	6.5	193.5	4.4	181.2	10.(
DJ	159.4	5.3	174.8	5.8	183.5	1.8	175.1	4.8
CJ	146.8	8.0	170.1	8.1	190.0	3.5	155.5	6.(
EJ	172.9	4.7	195.1	14.8	206.7	3.6	193.0	6.1
RL	173.6	5.6	218.6	3.3	207.3	2.7	213.7	6.1
SM	124.7	6.0	170.3	6.4	185.2	3.1	158.3	6.3
AM	139.3	4.8	177.6	2.4	183.0	1.6	161.4	5.1
GM	183.1	9.3	190.0	8.8	205.0	4.2	218.0	7.
PN	194.8	5.6	212.5	5.0	203.8	2.9	198.7	6.(
BP	138.3	4.2	181.2	4.7	184.6	2.9	162.9	5.
AP	147.1	9.0	188.6	6.7	201.5	3.1	177.3	9.
JS	166.3	2.6	183.5	5.4	192.1	1.4	179.1	4.
MS	158.1	4.1	178.7	7.5	184.3	2.0	174.6	8.
GT	138.0	6.4	171.9	5.1	188.4	4.1	162.6	6.
MT	186.4	8.7	193.4	4.6	210.5	4.8	210.2	8.
ET	155.5	5.0	199.5	3.8	200.5	1.8	191.8	5.
MTH	174.1	4.7	193.1	8.9	203.1	3.1	186.6	6.

ɔle 60 to 64 . The statistical analaysis of the skinfold thickne for the five sites employed at Halley Bay, employ analysis of variance techniques

ɔle 60 . Site I - Pectoral.

ırce of Variation	Sum of Squares	Degrees of Freedom	Mean Square	Var Ra
;ween Men	100,297.0	17	5,899.82	159
;ween Months	4,396.0	11	399.64	10
;idual	7,026.0	187	37.57	-
;al	111,719.0	215	-	-

ɔle 61 . Site II - Lateral Arm.

ırce of Variation	Sum of Squares	Degrees of Freedom	Mean Squares	Var Ra
tween Men	45,671.0	17	2,686.5	8
tween Months	1,507.0	11	137.0	
;idual	6,054.0	187	32.37	
tal	53,322.0	215	-	

le 62. Site III - Scapula

rce of Variation	Sum of Squares	Degrees of Freedom	Mean Square	Vari Ra
ween Men	22,201.0	17	1,305.9	19
ween Months	1,383.0	11	125.73	1
idual	1,229.0	187	6.572	-
al	24,813.0	215	-	-

le 63. Site IV - Abdominal

rce of Variation	Sum of Squares	Degrees of Freedom	Mean Square	Var R
ween Men	62,292.0	17	3,664.2	9
ween Months	2,527.0	11	229.7	
idual	6,971.0	187	37.27	
al	71,790.0	215	-	

ble 64 . Site V - Lateral side of thigh

urce of Variation	Sum of Squares	Degrees of Freedom	Mean Square	V
tween Men	88,235.0	16	5,514.68	
tween Months	716.0	11	65.09	
sidual	108,563.0	176	616.83	
tal	197,514.0	203	-	

Table 65. Results of Diet Survey - First Period

Name	Date 1961	Protein g	Fat g	CHO g	Total cal
DE	13.2	118.5	117.3	383.1	2989.3
MJ	13.2	118.5	124.7	396.4	3060.4
JS	16.2	124.9	144.9	464.1	3743.2
ED	16.2	183.3	193.6	682.8	5003.6
DED	22.2	234.3	191.8	692.7	5368.9
SM	22.2	227.2	125.4	650.0	4502.7
GM	22.2	143.9	46.3	292.4	2138.9
MBR	26.2	106.5	184.3	455.3	3848.3
PN	26.2	147.4	202.6	341.2	3735.8
*GM	26.2	157.5	112.9	201.6	2179.7
CD	28.2	151.6	149.3	449.9	3677.1
RL	28.2	147.8	212.9	578.7	4756.8
MTH	2.3	163.4	289.0	630.1	5721.5
DA	2.3	97.5	177.6	494.4	4087.3
AP	8.3	93.2	168.7	581.9	4209.6
GB	8.3	104.1	205.3	560.4	4432.2
MS	10.3	140.1	158.5	424.0	3633.9
MB	10.3	150.9	119.6	330.9	2985.0
GT	14.3	124.2	185.3	495.4	4087.0
DJ	14.	120.4	179.3	440.8	3773.2
AM	16.3	107.7	166.3	486.5	3780.9
CJ	16.3	68.3	98.0	502.1	3202.9
EJ	22.3	73.1	103.7	386.8	2758.6
BP	22.3	102.6	89.8	444.7	3101.6
MT	26.3	141.8	128.3	383.6	3189.5
ET	26.3	127.4	172.7	450.3	3817.8

* Not employed in any calculations.

Table 66. Results of Diet Survey - Second Period

Name	Date 1961	Protein g	Fat g	CHO g	Total cal
PN	29.3	87.4	172.8	499.0	3787.8
MTH	29.3	172.6	389.3	735.5	6858.7
SM	30.3	187.3	162.0	499.0	4151.5
ED	30.3	267.7	156.1	448.0	4224.1
DED	5.4	167.3	216.4	701.8	5197.2
MBR	5.4	76.8	125.5	354.7	2851.9
MJ	11.4	169.0	142.2	436.3	3728.5
JS	11.4	135.5	111.7	396.3	3085.4
RL	14.4	122.2	209.1	395.4	4182.4
GB	14.4	162.7	233.9	687.1	5567.8
CD	18.4	107.3	143.1	397.8	3402.0
DE	18.4	143.4	192.6	551.5	4574.9
AP	21.4	108.0	240.3	460.0	4506.6
DA	21.4	90.6	224.6	549.3	4573.7
MB	25.4	85.2	112.3	484.2	3373.5
MS	25.4	74.9	92.9	414.4	2824.1
AM	28.4	118.7	165.5	703.7	4857.1
CJ	28.4	124.9	145.2	665.7	4129.2
BP	2.5	171.9	202.5	480.2	4639.9
EJ	2.5	68.9	108.7	245.5	2398.9
GT	5.5	118.0	122.9	347.4	2922.1
MT	5.5	80.3	70.0	318.7	2253.5
ET	9.5	83.9	100.9	343.3	2566.5
DJ	9.5	78.2	98.6	417.3	2867.4
GM	9.5	105.6	141.6	336.4	3026.4

Table 67. Results of Diet Survey - Third Period

Name	Date 1961	Protein g	Fat g	CHO g	Total cal
PN	12.5	129.9	229.5	434.1	4417.5
SM	12.5	136.3	176.1	539.5	4304.0
MBR	16.5	61.8	86.0	289.9	2133.6
ED	16.5	86.7	127.2	397.3	3019.8
JS	18.5	90.3	175.7	539.0	4036.2
DE	18.5	103.0	170.8	510.7	3937.3
MJ	18.5	58.4	140.9	384.0	3089.5
CD	18.5	122.2	176.2	539.2	4234.0
DED	23.5	119.3	137.0	641.5	4303.2
RL	23.5	126.7	180.6	495.7	4026.0
DA	26.5	102.4	230.0	487.7	4175.5
AP	26.5	94.0	133.4	380.7	3094.0
CJ	30.5	100.0	112.4	333.6	2968.5
MTH	30.5	150.8	266.2	590.2	5454.7
AM	2.6	112.5	148.8	583.5	3948.1
MB	2.6	76.4	124.0	331.0	2630.7
MS	6.8	92.9	123.5	344.9	2813.9
GB	6.6	101.5	143.4	461.2	3440.9
EJ	9.6	53.7	67.5	260.7	1848.2
BP	9.6	91.9	88.2	443.2	2880.7
GT	12.6	133.9	144.5	333.9	3152.7
MT	13.6	82.4	115.0	374.9	2790.3
DJ	16.6	74.6	129.7	438.2	3175.1
ET	16.6	81.3	118.0	452.0	3128.3
GM	16.6	65.6	85.9	447.6	2954.6

Table 68. Results of Diet Survey - Fourth Period

Name	Date 1961	Protein g	Fat g	CHO g	Total cal
MBR	25.6	81.7	138.9	473.0	3312.0
PN	25.6	124.7	145.9	361.9	3276.4
SM	25.6	94.6	114.3	416.0	3012.5
ED	27.6	97.3	151.6	509.3	3705.6
DED	27.6	127.5	209.0	879.3	5741.2
JS	29.6	121.0	204.8	471.9	4130.9
CD	29.6	125.1	155.8	358.0	3202.2
MJ	4.7	96.4	121.6	631.1	3804.4
DA	4.7	131.1	152.3	533.9	3953.4
DE	7.7	60.9	110.4	413.3	2791.8
MTH	7.7	51.0	91.5	373.1	2449.9
AP	11.7	73.4	118.9	488.3	3049.9
RL	11.7	131.1	186.4	658.8	4957.9
CJ	14.7	108.3	150.8	531.9	3864.9
AM	14.7	85.9	120.9	507.1	3379.4
GB	18.7	79.3	148.6	567.6	3857.9
MB	18.7	78.0	115.3	472.2	3233.7
MS	21.7	62.8	97.1	404.3	2716.2
BP	21.7	64.4	102.0	355.5	2621.2
EJ	25.7	78.4	122.9	423.5	3173.3
MT	25.7	88.6	157.8	263.5	2672.2
GT	28.7	100.5	160.0	348.5	3329.2
DJ	28.7	90.5	121.5	495.6	3527.1
ET	1.8	49.8	62.1	234.5	1633.6
GM	1.8	57.6	92.5	310.8	2256.9

Table 69. Results of Diet Survey - Fifth Period

Name	Date 1961	Protein g	Fat g	CHO g	Total cal
SM	2.8	84.8	133.2	319.9	2710.2
PN	3.8	106.5	176.9	314.8	3128.0
MTH	6.8	80.9	151.6	251.9	2686.9
MBR	8.8	52.8	76.8	343.2	2260.5
ED	8.8	96.0	126.3	372.8	2989.1
CD	11.8	87.3	101.8	299.2	2691.9
DED	11.8	125.2	216.4	803.7	5548.5
MJ	15.8	136.7	171.7	486.8	4288.9
JS	15.8	98.5	112.0	371.3	2847.9
DA	18.8	85.9	183.7	353.2	3177.7
DE	18.8	81.9	221.6	388.7	3737.1
CJ	22.8	118.5	150.3	365.2	3221.2
AP	22.8	73.8	95.9	429.4	2729.4
MS	25.8	81.8	137.1	407.5	3084.2
MB	25.8	110.0	178.5	280.9	3334.7
RL	29.8	146.0	282.8	617.3	5445.9
MT	29.8	51.7	84.2	275.3	2054.3
BP	1.9	88.2	218.6	645.5	4770.1
AM	1.9	99.5	272.1	409.9	4398.8
GT	5.9	95.7	163.8	402.9	3379.6
GB	5.9	103.7	107.5	433.9	3153.3
EJ	7.9	79.5	91.8	336.0	2439.2
DJ	7.9	88.4	114.2	419.2	2987.7
ET	11.9	69.3	154.7	506.2	3546.6
GM	11.9	45.4	112.8	258.9	2138.5

Table 70. Results of Diet Survey - Sixth Period

Name	Date 1961	Protein g	Fat g	CHO g	Total Cals.
SM	15.9	115.4	174.6	536.9	4097.5
PN	15.9	98.6	148.3	441.5	3869.0
JS	9.9	96.8	169.3	450.9	3829.8
MBR	9.9	70.2	135.9	413.6	3105.6
CD	23.9	63.7	115.5	338.9	2871.7
DE	23.9	79.9	151.6	385.1	3200.3
MJ	23.9	72.0	128.8	354.9	2849.7
AP	26.9	91.2	151.7	539.1	3868.5
CJ	26.9	138.8	269.6	787.3	6415.8
DA	26.9	93.5	211.3	505.9	4387.6
MB	29.9	78.8	151.5	384.1	3182.7
ED	29.9	82.8	152.9	481.6	3469.7
GT	29.9	110.2	152.1	387.5	3340.2
RL	3.10	134.0	336.8	652.0	6131.5
MT	3.10	109.5	168.6	472.5	3859.8
DED	3.10	123.9	232.0	783.0	5611.1
AM	5.10	89.7	142.0	529.2	3803.6
BP	5.10	94.7	99.9	508.4	3444.4
GB	5.10	106.8	129.6	459.5	3372.0
ET	9.10	24.9	92.1	221.5	1791.5
DJ	9. 10	69.6	108.8	440.8	2986.5
EJ	9.10	79.8	117.3	478.0	3323.0
MTH	13.10	106.2	207.2	514.5	4334.1
GM	13.10	63.7	101.2	264.6	2193.5
MS	13.10	101.1	218.5	386.1	3888.0

Table 71. Results of Diet Survey - Seventh Period.

Name	Date 1961	Protein g	Fat g	CHO g	Total cal
CJ	13.10	117.3	189.8	556.5	4420.7
DA	15.10	85.5	130.9	446.6	3258.4
MB	3.11	76.7	84.8	564.8	3291.0
DED	3.11	73.7	114.3	813.4	4681.4
ET	3.11	69.3	123.5	522.0	3552.6
ED	3.11	62.1	81.6	405.6	2692.2
PN	6.11	103.5	159.6	352.0	3294.5
SM	6.11	87.7	117.2	298.9	2500.2
MBR	6.11	83.2	145.7	461.2	3371.2
CD	8.11	104.5	169.6	326.5	3253.6
JS	8.11	69.4	110.5	282.4	2341.8
MJ	8.11	103.6	173.2	602.8	4238.2
AP	14.11	73.7	157.5	392.4	3316.6
MTH	14.11	108.8	249.5	488.8	4705.9
DE	14.11	96.6	214.4	423.9	3932.3
RL	16.11	191.7	351.1	692.6	6620.0
MT	16.11	131.8	119.2	416.8	3195.8
MS	16.11	97.4	145.0	440.1	3395.7
BP	20.11	103.7	106.0	449.2	3128.0
AM	20.11	124.2	156.2	369.5	3357.1
GB	20.11	94.7	156.9	502.5	3728.4
EJ	22.11	78.2	100.2	361.6	2669.4
GT	22.11	100.6	136.6	419.0	3370.1
GM	22.11	106.3	129.5	385.2	3076.5
DJ	22.11	86.7	108.6	367.4	2781.9

Table 72. Results of Diet Survey - Eighth Period

Name	Date 1961	Protein g	Fat g	CHO g	Total cal
PN	30.11	102.7	156.3	369.2	3324.8
MBR	30.11	83.8	121.1	437.1	3116.0
SM	30.11	132.4	141.8	417.8	3431.1
DE	5.12	111.3	149.1	450.0	3553.8
CD	5.12	98.1	174.6	353.7	3436.1
JS	5.12	86.6	148.6	388.1	3200.3
ED	13.12	78.1	189.4	488.4	3923.7
MTH	13.12	95.1	259.5	530.8	4791.9
DED	13.12	115.0	242.4	765.8	5730.4
MS	15.12	133.3	180.8	481.8	4139.9
DA	15.12	128.0	190.3	451.7	4089.1
AP	15.12	134.8	159.5	417.5	3610.5
GB	18.12	182.7	294.0	645.2	5908.0
MJ	18.12	141.0	187.6	442.1	4014.5
MT	18.12	103.9	151.2	384.6	3275.0
DJ	21.12	84.5	151.0	437.0	3720.0
RL	21.12	114.2	252.6	543.7	4856.8
BP	21.12	87.3	156.2	411.7	3385.3
AM	21.12	77.5	117.2	355.2	2746.7
EJ	21.12	87.8	143.2	390.5	3384.5
CJ	27.12	83.2	149.5	364.6	3155.4
GT	27.12	88.5	163.8	370.4	3277.4
MB	27.12	80.1	127.0	339.9	2873.0
ET	27.12	65.2	154.1	299.8	2792.0
GM	27.12	88.1	171.5	426.8	3586.5

Table 73. Figures of total calorie intakes for each period of the diet survey

Subject	Period I	II	III	IV	V	VI	VII	VIII
DA	4087.3	4773.7	4175.5	3953.4	3177.7	4387.6	3258.4	4089.1
MB	2985.0	3373.5	2630.7	3233.7	3334.7	3182.7	3291.0	2873.0
GB	4432.2	5567.8	3440.9	3857.9	3153.3	3372.0	3728.4	5908.0
MBR	3848.3	2851.9	2133.6	3312.0	2260.5	3105.6	3371.2	3116.0
CD	3677.1	3402.0	4234.0	3202.2	2691.9	2871.7	3253.6	3436.1
ED	5003.6	4224.1	3019.8	3705.6	2989.1	3469.7	2692.2	3923.7
DE	2989.3	4574.9	3937.3	2791.8	3737.1	3200.3	3932.3	3553.8
DED	5368.9	5197.2	4197.2	5741.2	5548.5	5611.1	4681.4	5730.5
MJ	3060.4	3728.5	3089.5	3804.4	4288.9	2849.7	4238.2	4014.5
DJ	3773.2	2867.4	3175.1	3527.1	2987.7	2986.5	2781.9	3720.0
CJ	3202.9	4129.2	2968.5	3864.9	3221.2	6415.8	4420.7	3155.4
EJ	2758.6	2398.9	1848.2	3173.3	2439.2	3323.0	2669.4	3384.5
RL	4756.8	4182.4	4026.0	4957.9	5445.9	6131.5	6620.0	4856.8
SM	4502.7	4151.5	4304.0	3012.5	2710.2	4097.5	2500.2	3431.1
AM	3780.7	4857.1	3948.1	3379.4	4398.8	3803.6	3357.1	2746.7
GM	2138.9	3026.4	2954.6	2256.9	2138.5	2193.5	3076.5	3586.5
PN	3735.8	3787.8	4417.5	3276.4	3128.0	3869.0	3294.5	3324.8
BP	3101.6	4639.9	2880.7	2621.2	4770.1	3444.4	3128.0	3385.3
AP	4209.6	4616.6	3094.0	3049.9	2729.4	3868.5	3316.6	3610.5
JS	3743.2	3085.4	4036.2	4130.9	2847.9	3829.8	2341.8	3200.3
MS	3633.9	2824.1	2813.9	2716.2	3084.2	3888.0	3395.7	4139.9
GT	4087.0	2922.1	3152.7	3329.2	3379.6	3340.2	3370.1	3277.4
MT	3189.5	2253.5	2790.3	2672.2	2054.3	3859.8	3195.8	3275.0
ET	3817.8	2566.5	3128.3	1633.6	3546.6	1791.5	3552.6	2792.0
MTH	5721.5	6858.7	5454.7	2449.9	2686.9	4334.1	4705.9	4791.9

Table 74. The individual dietary protein consumption expressed as calories, for each period of the food intake survey.

Subject	Period I	II	III	IV	V	VI	VII	VIII
DA	390.0	400.0	409.6	524.0	343.6	374.0	342.0	512.0
MB	603.6	340.8	305.6	312.0	440.0	315.2	306.8	320.4
GB	416.4	650.8	406.0	317.2	414.8	427.2	378.8	730.8
MBR	426.0	307.2	247.2	326.8	211.2	280.8	332.8	335.2
CD	606.4	429.2	488.8	500.4	349.2	254.8	418.0	392.4
ED	613.2	1070.8	346.8	389.2	384.0	331.2	248.4	312.4
DE	474.0	573.6	412.0	243.6	327.6	319.6	386.4	445.2
DED	937.2	669.2	474.8	510.0	500.8	495.6	294.8	460.0
MJ	474.0	676.0	233.6	385.6	546.8	288.0	414.4	564.0
DJ	481.6	312.8	298.4	362.0	353.6	278.4	346.8	338.0
CJ	273.2	499.6	400.0	433.2	474.0	555.2	469.2	332.8
EJ	292.4	275.6	214.8	313.6	318.0	319.2	312.8	351.2
RL	591.2	488.8	506.8	524.4	584.0	536.0	766.8	456.8
SM	908.8	749.2	545.2	378.4	339.2	461.6	350.8	529.6
AM	430.8	474.8	450.0	343.6	398.0	358.8	496.8	310.0
GM	615.6	422.4	262.4	230.4	181.6	254.8	425.2	352.4
PN	589.6	349.6	519.6	498.8	426.0	394.4	414.0	410.8
BP	410.4	687.6	367.6	257.6	352.8	378.8	414.8	349.2
AP	372.8	480.0	376.0	293.6	295.2	364.8	294.8	539.2
JS	499.6	542.0	361.2	484.0	394.0	387.2	277.6	346.4
MS	560.4	299.6	371.6	251.2	327.2	404.4	389.6	533.2
GT	496.8	472.0	535.6	402.0	382.8	440.8	402.4	354.0
MT	567.2	322.0	329.6	354.4	206.8	438.0	527.2	415.6
ET	509.6	335.6	325.2	199.2	277.2	99.6	277.2	260.8
MTH	653.6	690.4	603.2	204.0	323.6	424.8	435.2	380.4

Table 75 . The individual dietary fat consumption expressed as calories, for each period of the dietary intake survey.

Subject	Period I	II	III	IV	V	VI	VII	VIII
DA	1598.4	2111.4	2070.0	1370.7	1653.3	1901.7	1178.1	1712.7
MB	1076.4	1010.7	1116.0	1037.7	1606.5	1363.5	763.2	1143.0
GB	1847.7	2105.1	1290.6	1337.4	967.5	1166.4	1412.1	2646.0
MBR	1658.7	1129.5	774.0	1250.1	691.2	1223.1	1311.3	1089.9
CD	1343.7	1287.9	1585.8	1402.2	916.2	1039.5	1526.4	1571.4
ED	1742.4	1404.9	1144.8	1364.4	1136.7	1376.1	734.4	1704.6
DE	1055.7	1733.4	1537.2	993.6	1994.4	1364.4	1929.6	1341.9
DED	1726.2	1947.6	1233.0	1881.0	1947.6	2088.0	1028.7	2181.6
MJ	1122.3	1279.8	1268.1	1094.4	1545.3	1159.2	1558.8	1688.4
DJ	1613.7	869.4	1167.3	1093.5	1027.8	979.2	977.4	1359.0
CJ	882.0	1306.8	1011.6	1357.2	1352.7	2426.4	1708.2	1345.5
EJ	933.3	978.3	607.5	1106.1	826.2	1055.7	901.8	1288.8
RL	1916.1	1881.9	1625.4	1677.6	2545.2	3031.2	3159.9	2273.4
SM	1125.0	1458.0	1584.9	1028.7	1198.8	1571.4	1054.8	1276.2
AM	1496.7	1489.5	1339.2	1088.1	2448.9	1278.0	1405.8	1054.8
GM	416.7	1274.4	773.1	832.5	1015.2	910.8	1165.5	1543.5
PN	1823.4	1555.2	2065.5	1313.1	1586.7	1334.7	1436.4	1406.7
BP	808.2	1822.5	793.8	918.0	1967.4	899.1	954.0	1405.8
AP	1518.3	2250.0	1200.6	1070.1	863.1	1365.3	1417.5	1435.5
JS	1304.1	1005.3	1581.3	1843.2	1008.0	1523.7	994.5	1337.4
MS	1426.5	836.1	1111.5	873.9	1233.9	1966.5	1305.0	1627.2
GT	1667.7	1106.1	1300.5	1440.0	1474.2	1368.9	1229.4	1474.2
MT	1154.7	630.0	1035.0	1420.2	757.8	1517.4	1072.8	1360.8
ET	1554.3	908.1	1062.0	558.9	1392.3	828.9	1111.5	1386.9
MTH	2601.0	3503.7	2395.8	823.5	1364.4	1864.8	2245.5	2335.5

Table 76. The individual dietary carbohydrate consumption as calories, for each period of the food intake survey.

Subject	Period I	II	III	IV	V	VI	VII	VIII
DA	1829.3	2069.4	1804.5	1975.4	1306.8	1871.8	1652.4	1671.3
MB	1224.3	1791.5	1224.7	1747.1	1039.3	1421.2	2089.8	1257.6
GB	2073.5	2542.3	1706.4	2100.1	1605.4	1700.2	1859.3	2387.2
MBR	1684.6	1312.4	1072.6	1750.1	1272.1	1530.3	1706.4	1617.3
CD	1664.6	1471.9	1995.0	1324.6	1107.0	1253.9	1208.1	1308.7
ED	2526.4	1657.6	1470.0	1884.4	1379.4	1781.9	1500.7	1807.1
DE	1379.2	2040.6	1889.6	1529.2	1438.2	1424.9	1568.4	1665.0
DED	2563.0	2596.7	2362.8	3253.4	2973.7	2897.1	3009.6	2833.5
MJ	1466.7	1614.3	1420.8	2335.1	1801.2	1313.1	2230.4	1635.8
DJ	1631.0	1544.0	1621.3	1833.7	1551.0	1631.0	1359.4	1616.9
CJ	1857.8	2463.1	1234.3	1968.0	1351.2	2913.0	2059.1	1349.0
EJ	1431.2	908.4	964.6	1567.0	1243.2	1768.6	1337.9	1444.9
RL	2141.2	1463.0	1834.1	2437.6	2284.0	2412.4	2562.6	2011.7
SM	2405.0	1846.3	1996.2	1539.2	1183.6	1986.5	1105.9	1545.9
AM	1800.1	2603.7	2159.0	1876.3	1516.6	1958.0	1367.2	1314.2
GM	1082.0	1244.7	1656.1	1150.0	957.9	979.0	1425.2	1579.2
PN	1262.4	1846.3	1606.2	1339.0	1164.8	1633.6	1302.4	1366.0
BP	1645.4	1776.7	1639.8	1315.4	2388.4	1881.1	1662.0	1523.3
AP	2153.0	1739.0	1408.6	1806.7	1588.8	1994.7	1451.9	1544.8
JS	1717.2	1466.3	1994.3	1746.0	1373.8	1668.3	1044.9	1436.0
MS	1568.8	1533.3	1276.1	1495.9	1507.8	1428.6	1628.4	1782.7
GT	1833.0	1285.4	1235.4	1289.5	1490.7	1433.8	1550.3	1370.5
MT	1419.3	1179.2	1387.1	975.0	1018.6	1748.3	1542.2	1423.0
ET	1666.1	1270.2	1672.4	867.7	1872.9	819.6	1931.4	1109.3
MTH	2331.4	2721.4	2183.7	1380.5	932.0	1903.7	1808.6	1964.0

Table. 77. The individual mean levels for the year of protein, fat and carbohydrate dietary intakes, with standard deviations expressed as calories.

Name	Protein	SD	Fat	SD	Carbohydrate	SD
DA	412.0	69.7	1699.5	327.1	1772.5	229.3
MB	368.1	105.2	1139.8	249.3	1474.5	358.1
GB	467.8	142.7	1596.6	557.2	1996.6	338.4
MBR	316.3	69.8	1141.0	254.4	1493.1	245.9
CD	429.8	104.4	1334.1	246.2	1416.8	302.0
ED	461.9	239.9	1326.0	325.6	1750.8	359.0
DE	397.9	102.3	1493.6	372.8	1616.6	241.0
DED	542.9	187.8	1754.4	412.3	2811.3	290.0
MJ	447.9	150.5	1339.4	229.3	1727.1	374.2
DJ	346.5	118.4	1135.8	241.5	1598.5	123.8
CJ	429.6	97.0	1423.8	472.9	1899.3	586.6
EJ	299.8	38.1	962.3	202.5	1333.2	290.5
RL	556.9	95.0	2263.7	593.3	2143.3	116.2
SM	532.9	202.2	1287.3	225.6	1701.1	425.5
AM	407.9	66.4	1450.2	436.0	1824.3	431.5
GM	343.0	141.3	991.6	341.2	1259.2	267.9
PN	450.5	75.5	1565.2	261.7	1440.0	229.8
BP	402.5	122.9	1196.1	472.4	1728.8	313.0
AP	377.1	89.4	1390.1	410.9	1711.1	264.0
JS	411.5	86.3	1324.6	311.1	1555.8	287.1
MS	392.1	108.0	1297.6	375.1	1527.8	143.5
GT	435.9	59.7	1382.5	176.9	1436.1	194.0
MT	395.1	115.9	1118.6	202.5	1336.5	259.5
ET	285.6	118.7	1100.4	333.9	1401.1	439.9
MTH	464.4	170.9	2142.0	809.0	1903.2	554.7

Table 78 to 81. The statistical analysis for seasonal changes in food intake, using the analysis of variance employing three unequal blocks.

Table 78. Total Calories

Source of Variation	Sum of squares	Degrees of Freedom	Mean Square	Variance Ratio	Significant at
Between Men	88,150,980.2	24	3,672,950.8	8.3718	0.1% level
Between Seasons	8,993,550.0	2	4,496,775.0	10.2496	0.1% level
Residual	54,840,540.0	125	438,724.0	-	-

Table 79. Fat Calories

Source of Variation	Sum of squares	Degrees of Freedom	Mean Square	Variance Ratio	Significant at
Between Men	21,079,305.5	24	878,304.3	11.21	0.1% level
Between Seasons	1,235,657.0	2	617,820.0	7.885	0.1% level
Residual	744,671.0	125	78,357.5		

Table 80. Protein Calories

Source of Variation	Sum of squares	Degreesof Freedom	Mean Square	Variance Ratio	Significant at
Between Men	307,982.0	24	12,833.0	0.85	NS
Between Seasons	916,756.0	2	458,378.0	30.47	0.1% level
Residual	1,880,085.0	125	15,040.6		

Table 81. Carbohydrate Calories

Source of Variation	Sum of squares	Degrees of Freedom	Mean Square	Variance Ratio	Significant at
Between Men	20,094,283.0	24	837,262.0	9.669	0.1% level
Between Seasons	811,500.0	2	405,750.0	4.686	1.0% level
Residual	10,823,808.0	125	86,590.0		

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Table 82. The individual assessments of blood pressure (mm Hg) and of pulse rate (beats/minute) at monthly intervals during the year.

Subject	Feb		Mar		Apr		May	
	B.P. syst./diast.	Pulse rate	B.P. syst./diast.	Pulse rate	B.P. syst./diast.	Pulse rate	B.P. syst./diast.	Pulse rate
DJ	95/70	56	90/60	64	110/70	50	110/80	56
AM	-	-	110/80	52	110/80	50	100/75	58
ET	130/70	64	-	-	120/75	62	130/75	71
MS	110/75	66	100/70	68	90/70	50	106/70	64
GT	130/80	58	110/80	58	110/80	60	120/90	64
CJ	110/70	52	120/80	52	90/70	52	110/70	48
RL	-	-	115/80	76	110/70	78	120/85	68
JS	110/70	58	120/80	56	100/60	62	90/65	60
AP	-	-	-	-	110/70	46	110/80	52
DED	110/70	52	110/75	60	110/75	54	90/55	58
MJ	-	-	105/80	62	110/80	80	110/80	74
DA	110/70	68	126/75	62	110/65	72	90/60	60
GB	135/85	50	120/90	52	110/80	54	110/80	52
MB	110/80	54	90/60	52	90/70	46	95/80	50
PN	110/60	52	100/60	62	100/70	50	90/60	54
BP	100/70	64	100/70	64	130/80	56	90/50	54
SM	90/40	58	90/50	52	120/40	60	90/60	52
GM	-	-	100/70	56	120/90	58	110/80	62

Subject	Feb BP. syst/diast	Feb Pulse rate	Mar B.P. syst/diast	Mar Pulse rate	Apr B.P. syst/diast	Apr Pulse rate	May B.P. syst/diast	May Pulse rate
CD	-	-	110/80	54	110/80	54	120/80	58
ED	90/60	62	110/80	52	120/80	62	105/65	54
MBR	90/70	54	-	-	-	64	110/80	70
MT	110/70	64	110/70	54	110/70	68	140/95	66
MTH	110/70	48	110/65	58	110/70	56	100/70	56

	Jun		July		Aug		Sept	
DJ	95/62	68	94/60	56	95/65	54	110/80	56
AM	115/80	48	90/65	50	-	-	110/80	48
ET	120/80	60	125/80	54	130/100	52	120/90	58
MS	100/60	54	120/70	54	90/70	56	115/80	60
GT	125/80	60	120/75	60	115/70	52	120/80	54
CJ	85/70	52	115/80	54	85/65	56	105/75	52
RL	130/90	66	130/80	68	130/90	76	120/85	79
JS	90/55	70	80/50	60	110/70	56	110/70	64
AP	95/70	54	90/60	56	110/85	46	110/80	52
DED	95/60	58	100/70	50	95/65	56	125/90	58

Subject	June B.P. syst/diast	June Pulse rate	July B.P. syst/diast	July Pulse rate	Aug B.P. syst/diast	Aug Pulse rate	Sept B.P. syst/diast	Sept Pulse rate
MJ	100/75	66	110/80	56	110/80	64	115/70	64
DA	100/60	68	110/80	62	110/70	68	100/70	64
GB	120/90	54	110/80	50	125/75	54	90/70	46
MB	110/80	48	100/70	50	115/70	52	100/75	57
PN	90/60	60	105/75	56	95/65	50	120/70	58
BP	100/70	50	-	-	120/80	52	100/60	64
SM	-	-	80/60	56	105/75	60	120/60	64
GM	95/70	54	85/60	56	90/70	66	110/90	52
CD	130/90	62	-	-	130/90	56	130/90	54
ED	110/80	52	120/80	50	95/65	50	110/70	58
MBR	95/70	50	110/70	56	105/70	56	100/70	54
MT	100/60	66	100/60	64	110/80	64	115/70	66
MTH	110/80	54	135/75	58	-	-	120/70	74

	Oct		Nov		Dec		Jan '62	
DJ	100/65	48	105/70	56	100/70	60	100/80	50
AM	130/100	52	125/90	60	100/65	50	110/80	54
ET	120/70	58	-	-	135/80	61	140/90	62
MS	95/60	60	105/70	52	110/70	53	80/50	60

Subject	Oct B.P. syst / diast	Pulse rate	Nov B.P. syst / diast	Pulse rate	Dec B.P. syst / diast	Pulse rate	Jan '62 B.P. syst / diast	Pulse rate
GT	-	-	120/90	48	120/75	29	110/80	54
CJ	125/90	56	-	-	-	-	115/75	50
RL	120/80	64	125/90	74	-	-	145/100	72
JS	115/80	54	115/70	58	110/75	54	120/80	58
AP	120/65	52	115/75	46	90/60	46	115/80	42
DED	110/80	56	-	-	110/70	50	100/60	52
MJ	115/75	60	-	-	135/85	66	115/75	66
DA	95/70	60	-	-	-	-	125/80	62
GB	100/70	50	125/90	56	110/80	46	110/65	72
MB	100/70	54	-	-	105/60	54	110/70	48
PN	90/55	54	125/75	68	80/60	54	115/60	52
BP	90/55	52	110/70	54	110/80	60	125/80	52
SM	-	-	105/76	54	110/70	62	80/50	56
GM	100/75	64	85/55	54	90/65	55	110/70	60
CD	125/90	56	130/90	70	120/75	62	-	-
ED	120/70	60	-	-	120/70	59	105/65	54
MBR	110/70	54	110/65	60	100/80	55	100/55	42
MT	105/75	64	120/80	64	130/85	64	120/80	64
MTH	-	-	135/80	56	110/55	60	110/60	54

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Table 83. The individual values of serum total lipids at monthly intervals, expressed as mg/100 ml serum.

Subjects	Feb	Mar	Apr	May	Jne	July	Aug	Sept	Oct	Nov	Dec	Jan
SM	655		596	595		627	646	560	620	577	549	620
DA	802		901	787	802	712	725	693	726		828	778
AP	906		713	767	732	737	754	703	707	682	720	700
MBR	641	627	592	593	620	613	614	594	571	598	614	592
GB	699	806	848	828	669	690	739	808	687	668	820	798
CJ	625		612		558	611	591	595	603		645	560
MS	602	529	549	524	571	538	577	558	468	490	457	565
RL	1133	892	806	876	869	957	958	960	950	927	828	901
MJ	863	756	756	794	813	816	780	700	712	744	761	701
GT	681	722	636	600	620	696	634	655	724	629	662	648
DED	528	556	624	625	576	658	635	575	681		561	547
PN	638	584	627	579	616	610	552	557	588	570	570	608
CD	815	655	700	977	777	664	1038	718	1029	776	1023	1084
ET	591	561	477	455	474	453	435	480	447		562	480
MTH	663	703	728	699	763	733	715	761		689	673	731
GM	616	598	602	595	596	618	618	677	675	504	588	613
MT	652	712	655	619	643	686	683	540	617	627	646	598
DJ	769	608	558	589	598	621	613	550	596	551	594	604
AM	558	528	503	478	476	476	528	504	513	477	454	496
DE	657	686	724	765	813	787	796	791	675	665	665	628
ED	591	575	597	519	651	599	557	553	543		600	533
MB	559	528	578	544	548	592	525	539	539		592	593
BP	769	769	773	563	851	871	751	692	766	713	739	732
JS	597	632	678	645	654	616	682	582	584	634	584	494

Table 84. The individual values of serum total fatty acids at monthly intervals, expressed as mg/100 ml serum.

Subject	Feb	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan
SM	388		345	346		373	401	332	374	333	320	369
DA	496		542	493	518	437	449	420	465		519	490
AP	500		400	424	404	407	427	389	402	377	400	403
MBR	352	350	328	342	342	344	360	334	330	338	350	338
GB	386	478	512	535	392	406	430	502	386	366	514	566
CJ	351		334		320	344	332	334	333		370	322
MS	363	305	321	286	338	300	297	328	261	278	255	330
RL	670	528	464	504	498	570	580	478	548	567	468	522
MJ	465	464	464	512	525	525	492	434	438	461	480	434
GT	401	424	356	337	357	385	351	368	413	351	372	374
DED	315	328	372	400	354	397	378	350	372		334	328
PN	352	319	339	318	330	330	297	313	328	316	323	350
CD	534	396	416	690	502	422	734	464	744	507	745	768
ET	336	319	273	238	273	259	245	273	246		296	277
MTH	371	397	416	412	457	436	411	429		379	375	417
GM	355	348	336	336	340	352	352	398	397	281	340	383
MT	358	394	359	332	350	383	383	298	332	330	362	326
DJ	470	362	329	340	350	368	368	316	346	316	346	340
AM	320	307	292	264	268	261	298	280	282	256	248	277
DE	346	372	392	418	451	439	434	428	371	370	372	364
ED	334	324	339	288	365	334	308	304	293		331	297
MB	318	296	286	306	303	336	280	304	304		350	334
BP	446	446	446	470	516	523	435	404	440	466	421	421
JS	330	352	385	359	367	331	342	324	327	363	332	280

Table 85. The individual values of serum phospholipids at monthly intervals, expressed in mg/100 ml serum

Subjects	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan
SM	212		211	194		206	202	178	201	193	181	202
DA	251		251	223	231	202	225	208	208		255	239
AP	329		285	293	274	275	288	262	280	276	272	282
MBR	199	201	196	197	215	199	205	208	192	206	214	219
GB	235	251	277	242	223	222	224	232	228	231	246	244
CJ	245		235		216	215	211	237	228		250	208
MS	208	206	199	210	212	197	190	211	178	187	182	203
RL	277	268	252	267	268	282	288	289	302	259	208	206
MJ	208	194	208	202	209	203	198	191	219	218	226	192
GT	248	276	237	218	230	267	240	243	259	247	256	253
DED	175	183	201	182	190	266	209	189	211		208	188
PN	236	227	237	217	230	227	206	210	216	204	196	215
CD	236	220	232	251	246	214	270	224	266	245	254	295
ET	192	201	187	190	182	173	166	187	185		244	179
MTH	223	245	250	240	245	242	233	266		242	237	259
GM	223	234	229	220	221	240	240	233	243	198	218	245
MT	240	250	236	230	236	249	245	238	243	251	243	239
DJ	273	221	198	200	200	211	216	204	221	211	220	224
AM	207	204	180	186	202	187	211	208	217	210	192	200
DE	239	258	292	289	326	284	303	294	255	240	242	237
ED	202	206	213	183	222	214	207	203	202		213	199
MB	204	212	221	212	230	232	222	228	230		224	237
BP	290	286	289	298	305	320	278	247	286	262	280	288
JS	215	218	234	219	230	224	209	203	208	196	210	195

Table 86. The individual serum total cholesterol levels at monthly intervals, expressed in mg/100 ml serum.

Subjects	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan
SM	196		182	184		184	175	168	178	180	169	182
DA	219		192	216	201	193	198	202	188		220	205
AP	300		222	249	240	242	245	230	214	216	232	206
MBR	223	211	199	186	208	207	186	192	178	192	194	182
GB	236	242	242	207	202	222	223	226	225	226	208	207
CJ	195		202	202	178	196	191	185	196		194	170
MS	170	158	162	171	162	174	157	161	149	151	144	168
RL	290	273	268	282	281	290	268	282	300	270	271	289
MJ	226	208	219	208	212	217	217	199	199	207	202	200
GT	198	207	204	192	188	225	206	208	226	198	207	192
DED	155	166	184	161	157	191	186	160	196		158	156
PN	210	192	212	191	213	208	189	187	190	188	182	187
CD	196	185	206	196	189	168	200	176	188	182	177	205
ET	192	176	143	158	142	137	137	146	142		190	144
MTH	219	225	229	208	223	216	226	246		231	220	229
MT	217	236	220	214	217	223	220	166	208	217	176	196
GM	188	174	181	186	184	187	179	201	198	159	204	210
DJ	207	173	164	183	181	183	173	167	177	167	176	192
AM	171	155	151	154	152	155	162	158	162	155	146	155
DE	235	234	240	254	257	254	264	268	224	216	214	186
ED	191	184	188	171	213	196	182	183	185		201	171
MB	176	164	161	169	172	181	168	161	170		168	182
BP	228	226	232	246	232	242	225	205	232	220	226	217
JS	197	208	206	214	212	214	194	192	189	196	183	151

Table 87 . The individual values of the cholesterol phospholipid ratio at monthly intervals.

Subjects	Feb	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan
SM	0.96		0.86	0.95		0.89	0.86	0.95	0.89	0.93	0.93	0.90
DA	0.87		0.77	0.97	0.87	0.96	0.88	0.97	0.90		0.86	0.86
AP	0.91		0.78	0.85	0.88	0.88	0.85	0.88	0.76	0.73	0.85	0.73
MBR	1.12	1.05	1.02	0.94	0.97	0.96	0.88	0.92	0.93	0.93	0.91	0.83
GB	1.0	0.96	0.87	0.86	0.91	1.00	1.00	0.97	0.99	0.98	0.85	0.85
CJ	0.80		0.86		0.82	0.91	0.86	0.76	0.86		0.78	0.82
MS	0.82	0.79	0.81	0.81	0.76	0.88	0.83	0.76	0.84	0.81	0.89	0.82
RL	1.05	1.02	1.06	1.06	1.05	1.03	0.93	0.98	0.99	0.96	1.07	1.09
MJ	1.09	1.07	1.05	1.03	1.01	1.07	0.91	1.04	0.91	0.95	0.90	1.07
GT	0.80	0.75	0.86	0.88	0.82	0.84	0.86	0.85	0.87	0.80	0.81	0.76
DED	0.89	0.91	0.92	0.88	0.83	0.93	0.89	0.85	0.93		0.76	0.83
PN	0.89	0.86	0.89	0.88	0.93	0.92	0.90	0.89	0.88	0.92	0.93	0.87
CD	0.83	0.84	0.89	0.76	0.77	0.79	0.74	0.79	0.71	0.74	0.70	0.70
ET	1.0	0.88	0.77	0.83	0.78	0.79	0.83	0.78	0.77		0.78	0.80
MTH	0.98	0.92	0.92	0.87	0.91	0.89	0.97	0.95		0.95	0.93	0.88
GM	0.84	0.74	0.79	0.84	0.83	0.78	0.75	0.87	0.81	0.80	0.81	0.86
MT	0.90	0.94	0.93	0.93	0.92	0.90	0.90	0.70	0.86	0.86	0.84	0.82
DJ	0.76	0.71	0.83	0.91	0.90	0.87	0.80	0.82	0.80	0.79	0.80	0.86
AM	0.83	0.76	0.84	0.83	0.75	0.83	0.77	0.76	0.75	0.74	0.76	0.77
DE	0.98	0.91	0.82	0.88	0.79	0.88	0.87	0.91	0.88	0.90	0.88	0.78
ED	0.95	0.89	0.88	0.94	0.96	0.92	0.88	0.90	0.92		0.95	0.86
MB	0.86	0.77	0.73	0.80	0.75	0.78	0.76	0.71	0.74		0.75	0.77
BP	0.79	0.79	0.80	0.82	0.76	0.76	0.81	0.83	0.81	0.84	0.81	0.75
JS	0.92	0.95	0.92	0.98	0.92	0.96	0.93	0.95	0.91	0.80	0.87	0.78

Table 88. The individual monthly values of serum free cholesterol (mg/100 ml serum)

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan
SM	37	-	43	40	-	45.	44	39	44	44	39	45
DA	50	-	48	43	48	43	46	52	49	-	58	49
AP	60	-	48	46	52	52	55	53	49	52	53	46
MBR	43	53	47	38	48	48	44	47	43	48	48	45
GB	46	57	54	40	44	46	53	62	52	54	47	42
CJ	44	-	50	54	45	45	46	52	52	-	52	44
MS	28	35	40	33	40	43	41	43	42	40	34	44
RL	67	70	70	70	72	89	74	76	82	72	74	73
MJ	49	51	54	50	54	59	54	52	52	57	57	54
GT	50	52	50	37	40	50	47	50	56	50	53	41
DED	26	37	43	33	34	44	42	43	46	-	36	36
PN	45	48	54	45	51	47	48	48	50	47	46	49
CD	48	50	51	54	51	51	62	52	65	55	58	64
ET	34	42	34	26	33	36	34	37	38	-	43	31
MTH	55	63	62	61	57	59	66	69	-	65	58	64
GM	45	45	48	48	51	49	51	59	54	48	44	54
MT	36	52	44	38	46	46	51	46	51	53	53	49
DJ	48	41	38	38	45	43	47	44	44	43	45	46
AM	34	36	35	33	37	39	44	42	41	36	35	38
DE	54	61	60	63	74	64	77	78	54	77	64	49
ED	41	46	45	40	55	45	48	51	50	-	47	41
MB	37	44	43	47	46	47	46	45	51	-	44	50
BP	53	54	61	64	68	62	62	57	65	60	59	59
JS	42	44	50	42	46	44	44	44	44	45	42	32

Table 89. The individual monthly values of serum cholesterol ester (mg/100 ml serum)

	Feb 1961	Mar	Apr	May	Jne	Jly	Aug	Sept	Oct	Nov	Dec	Jan 1962
SM	159		139	144	-	139	131	129	134	136	130	137
DA	169	-	144	173	153	150	152	150	139	-	162	156
AP	240	-	174	203	188	190	190	177	165	164	179	160
MBR	180	158	152	148	160	159	142	145	135	144	146	137
GB	190	185	188	167	158	176	170	191	173	172	161	165
CJ	151	-	152	148	133	151	145	133	144	-	142	126
MS	142	123	122	138	122	131	116	118	107	111	110	124
RL	223	203	198	212	209	201	194	206	218	198	197	216
MJ	177	157	165	158	158	158	163	147	147	150	145	146
GT	148	155	154	155	148	175	159	158	170	148	154	151
DED	129	129	141	128	123	147	144	117	150	-	122	120
PN	165	144	158	146	162	161	141	139	140	141	136	138
CD	148	135	155	136	138	117	138	124	123	127	119	141
ET	158	134	109	132	109	101	103	109	104	-	147	113
MTH	164	162	167	147	166	157	160	177	-	166	162	165
GM	143	129	133	138	133	138	128	142	144	169	132	156
MT	181	184	176	176	171	177	169	120	157	106	151	147
DJ	159	132	126	145	136	140	126	123	133	124	131	146
AM	137	119	116	121	115	116	118	116	121	119	111	117
DE	181	173	180	191	183	190	187	190	169	139	150	137
ED	150	138	143	131	158	151	134	132	135	-	154	130
MB	140	120	118	122	126	134	122	116	119	-	124	132
BP	175	172	171	182	164	180	163	148	167	160	167	158
JS	155	164	166	172	166	170	150	148	145	151	141	119

Table 90. The individual values of the serum beta-cholesterol levels at monthly intervals, expressed in mg/100 ml serum.

Subjects	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan
SM	140		142	146		144	142	139	141	152	132	142
DA	182		153	196	192	175	175	177	168		188	176
AP	232		156	186	189	200	212	176	162	163	182	127
MBR	194		167	165	197	183	175	170	152	169	176	168
GB	195		211	184	189	202	200	201	209	197	160	181
CJ	156		159	156	158	160	138	142	153		155	137
MS	121		128	148	147	144	140	139	126	124	114	138
RL	252		229	249	251	276	254	262	280	247	240	245
MJ	158		158	165	182	180	178	154	158	169	163	154
GT	173		169	150	157	180	166	178	188	160	186	159
DED	126			140	144	175	165	146	179		146	146
PN	171		174	155	167	178	154	155	154	156	148	161
CD	159		169	158	162	146	154	141	158		145	168
ET	156		112	118	113	116	111	105	115		139	108
MTH	179		172	171	172	166	172	194		194	182	184
GM	147		136	136	144	152	152	149	166	104	134	182
MT	188		194	188	196	205	183	119	188	197	174	182
DJ	157		113	141	140	136	154	120	129	109	138	141
AM	121		114	118	121	111	127	124	126	102	104	116
DE	196		171	226	239	213	234	237	190	182	171	134
ED	166		155	152	185	169	149	147	159		167	150
MB	129		116	133	132	143	135	115	114		100	122
BP	190		200	216	218	232	202	164	173	154	188	187
JS	169		190	188	192	199	177	172	169	163	148	123

Table 91. The individual values for the percentage of the serum total cholesterol on the beta-lipoprotein fraction at monthly intervals.

Subjects	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan
SM	67		67	71		69	72	70	70	77	71	67
DA	83		80	91	95	90	88	88	89		85	81
AP	77		70	75	79	83	86	77	76	76	78	62
MBR	82		84	89	94	88	94	89	85	89	91	92
GB	83		87	89	93	91	90	89	93	87	77	87
CJ	80		79	77	83	82	78	77	78		80	81
MS	71		79	87	91	83	89	86	85	82	79	82
RL	87		86	88	89	95	95	93	94	92	89	85
MJ	70		72	79		83	82	77	79	82	81	77
GT	87		83	78	84	80	81	86	83	81	90	83
DED	81			87	92	92	89	91	91		92	94
PN	81		82	81	78	86	82	83	81	83	81	86
CD	81		82	83	86	87	77	80	84		82	82
ET	81		78	75	79	85	81	72	81		73	75
MTH	82		75	82	77	77	76	79		64	83	80
GM	78		75	73	78	81	81	74	84	66	76	87
MT	87		88	88	90	92	83	72	90	91	85	93
DJ	76		69	77	77	74	89	72	73	65	78	73
AM	71		76	77	72	72	78	78	78	66	71	75
DE	83		71	89	93	84	89	89	85	84	80	72
ED	87		83	89	87	86	82	86	86		83	88
MB	73		72	79	77	79	80	71	66		60	67
BP	83		86	88	94	96	90	80	75	70	83	86
JS	86		88	88	90	93	90	90	90	83	81	82

Table 92. The individual serum alpha-cholesterol levels at monthly intervals, expressed in mg/100 ml serum.

Subjects	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan
SM	56		42	38		40	33	27	37	28	37	40
DA	37		39	20	9	18	23	25	20		32	29
AP	68		66	63	51	42	33	54	52	43	50	79
MBR	29		32	21	11	24	11	22	26	23	18	14
GB	41		31	23	13	20	23	25	16	29	48	26
CJ	39		43	46	20	36	53	43	43		39	33
MS	49		34	23	15	30	17	22	23	27	30	30
RL	38		39	33	36	14	14	20	20	23	31	44
MJ	68		59	43	30	37	61	45	41	62	39	46
GT	25		35	42	31	45	40	30	38	38	21	33
DED	29			21	18	16	21	14	17		12	10
PN	39		38	36	46	30	32	32	36	32	39	26
CD	37		37	32	27	22	46	35	30		32	37
ET	36		31	40	29	21	26	41	27		51	36
MTH	40		57	37	51	50	54	52		37	38	45
GM	41		45	50	40	35	27	52	32	55	42	28
MT	31		26	26	21	18	37	47	20	20	30	14
DJ	50		51	42	41	47	19	47	48	58	38	51
AM	50		36	36	31	44	35	34	36	53	42	39
DE	39		69	28	18	41	20	31	39	34	43	52
ED	25		33	19	28	27	33	36	26		34	21
MB	47		45	36	40	38	33	46	56		68	60
BP	38		32	30	14	10	23	41	59	66	38	30
JS	28		26	26	20	15	22	20	20	33	35	28

Table 93. The individual values of the serum triglycerides at monthly intervals, expressed in mg/100 ml serum.

Subjects	Feb	Mar	Apr	May	June	July	Aug	Sept	Oct	Nov	Dec	Jan
SM	131		101	112		135	173	126	143	105	104	136
DA	209		275	222	258	207	191	173	228		235	220
AP	102		79	77	81	81	82	82	92	70	85	95
MBR	88	100	86	102	80	95	119	88	102	95	99	91
GB	89	178	192	257	129	119	168	225	108	85	236	226
CJ	75		64		67	90	83	76	74		97	90
MS	120	81	99	42	108	71	85	100	63	71	51	103
RL	403	203	141	172	167	238	260	239	189	253	145	188
MJ	200	208	208	269	277	281	246	203	187	209	227	202
GT	127	126	83	77	94	76	72	89	115	76	87	93
DED	104	113	136	188	139	154	135	141	164		106	115
PN	71	60	63	64	55	57	54	58	80	75	93	105
CD	275	151	149	437	241	197	467	227	485	256	505	481
ET	92	86	67	11	70	69	57	67	44		21	74
MTH	101	115	127	144	174	160	139	126		95	98	122
GM	101	96	95	88	94	90	90	139	129	64	98	104
MT	63	92	71	47	65	85	95	48	51	39	89	56
DJ	173	118	104	100	118	125	132	89	101	83	102	82
AM	80	82	87	50	47	49	69	53	46	25	35	56
DE	51	70	61	83	96	105	93	90	79	102	99	112
ED	88	84	91	70	101	79	70	71	58		74	68
MB	77	65	50	54	54	81	46	65	65		110	78
BP	123	123	127	137	194	178	129	132	126	114	118	112
JS	72	86	107	87	91	54	93	79	81	98	88	61

Table 94. Individual lipid levels in serum taken from subjects before sledging expeditions. Lipid levels in mg/100 ml serum.

Subjects	Total Cholesterol	Phospho-lipids	Ratio C:P	Triglycer-ides	Total Fatty Acids	Total Lipids	Beta Chol-esterol	% Beta-Cholesterol	Alpha-Cholesterol
DA	219	251	0.87	209	496	802	182	83	37
	188	208	0.90	228	465	726	168	89	20
AP	300	329	0.91	102	500	906	232	77	68
	232	272	0.85	85	400	720	182	78	50
CJ	195	245	0.80	75	351	625	156	86	39
	191	211	0.86	83	332	591	148	78	43
DED	196	211	0.89	164	372	681	179	91	17
ET	142	185	0.77	44	246	447	115	81	27
GM	198	243	0.81	129	397	675	166	84	32
MT	220	245	0.90	95	383	683	183	83	37
DE	234	258	0.91	70	372	688	194	83	40
	224	255	0.88	79	371	675	190	85	34
	214	242	0.89	99	372	665	171	80	43
ED	185	202	0.92	58	293	543	159	86	26
MB	170	230	0.74	65	304	539	114	66	56
JS	183	210	0.87	88	332	584	148	81	35

Table 95 . Individual lipid levels in serum taken from subjects after sledging journeys. Lipid levels in mg/100 ml serum.

Subjects	Total Cholesterol	Phospho-lipids	Ratio C:P	Triglycer-ides	Total Fatty Acids	Total Lipids	Beta Chol-esterol	% Beta-Cholesterol	Alpha-Cholesterol
DA	192	251	0.77	275	542	901	153	80	39
	220	255	0.86	235	519	823	188	85	35
AP	222	285	0.78	79	400	713	156	70	66
	266	282	0.73	95	403	700	127	62	79
CJ	202	235	0.86	64	334	612	159	79	43
	185	237	0.78	76	334	595	142	77	43
DED	158	208	0.76	106	334	561	146	92	12
ET	190	244	0.78	21	296	562	139	73	51
GM	159	198	0.80	64	281	504	104	66	55
MT	166	238	0.70	48	298	540	119	72	47
DE	240	292	0.82	61	392	724	171	71	69
	216	240	0.90	102	370	665	182	84	34
	186	237	0.79	114	364	628	134	72	52
ED	201	213	0.94	74	331	600	167	83	34
MB	168	224	0.75	110	350	592	100	60	68
JS	151	195	0.77	61	280	494	123	82	28

Table 96. Individual lipid levels in serum taken from subjects one month after sledging expeditions. Lipid levels in kg/100 ml serum.

Subjects	Total Cholesterol	Phospho-lipids	Ratio C:P	Triglycer-ides	Total Fatty Acids	Total Lipids	Beta Chol-esterol	% Beta-Cholesterol	Alpha-Cholesterol
DA	216	223	0.97	222	493	787	196	91	20
	205	239	0.86	220	490	778	176	81	29
AP	249	293	0.82	77	424	767	186	75	63
CJ	202						156	77	46
	196	228	0.86	74	333	603	153	78	43
DED	156	188	0.83	115	328	547	146	94	10
ET	144	179	0.80	74	277	486	108	75	36
GM	176	218	0.81	98	340	588	134	76	42
MT	208	243	0.81	51	332	617	188	90	20
DE	254	289	0.88	83	418	765	226	87	28
	229	245	0.93	94	370	679	188	86	41
ED	171	199	0.86	68	297	533	150	88	21
MB	174	235	0.74	102	352	603	116	67	58

Tables 97 and 98. The estimated overall month and subject effects for serum total cholesterol and alpha and beta-cholesterol, and triglycerides.

Estimated parameter	Cholesterol				Triglycerides
	Total	Alpha	Beta	Beta %	
Residual variance	194	74	40.9	52.9	1900
Overall mean	199.6	33.8	165.8	82.3	123.05
Month Effects					
February	12.3	5.4	6.9	-2.0	7.2
April	.6	5.6	-5.0	-2.8	-11.3
May	2.1	-. 2	2.2	0.6	- .8
June	1.6	-6.6	8.1	2.9	-2.0
July	5.2	-5.0	10.2	2.9	-6.2
August	- 1.4	-5.5	4.0	2.7	7.8
September	- 4.8	1.3	-6.2	-0.9	-7.2
October	-2.6	- .7	-1.9	0.3	-1.6
December	-4.0	4.0	-8.0	-2.1	6.8
January	-8.9	1.7	-10.6	-1.5	7.3

Estimated subject effects	Cholesterol .				Triglycerides
	Total	Alpha	Beta	Beta %	
DA	3.8	-8.6	12.4	4.7	99
AP	38.4	22.0	16.4	-6.0	-37
MBR	-4.1	-13.0	8.9	6.5	-28
GB	20.2	-7.2	27.4	5.6	52
CJ/MJ *	-8.7	4.7	-13.4	-2.8	107
MS	-37.8	-6.5	-31.3	0.9	-39
RL	82.5	-5.5	88.0	7.8	91
GT	5.0	.2	4.8	1.2	-32
PN	-3.0	1.1	-4.1	-0.2	-53
CD	-10.1	-.3	-9.8	0.1	223
[illegible]	-46.5	0	-46.5	-4.3	-65
[illegible]	-10.6	5.4	-16.0	-3.6	-20
[illegible]	8.9	-7.0	15.9	4.5	-56
DJ	-19.3	9.6	-28.9	-6.5	-10
AM	-43.0	5.6	-48.6	-7.5	-65
DE	40.0	4.7	35.3	1.2	-36
ED	-11.5	-5.6	-5.9	3.4	-46
MB	-28.8	13.1	-41.9	9.9	-55
BP	28.9	-2.3	31.2	3.8	15
JS	-4.4	-10.8	6.4	5.5	-42

* CJ employed for Cholesterol responses and MJ for Triglyceride response