the protein of the tissues. This tissue breakdown leads to wasting. In diabetes wasting occurs because, though the food be sufficient, its glucose content cannot be oxidized; the fat and protein tissues are then called on. Their breakdown, while leading to the production of more sugar, also leads to more wasting of the body.

Lusk states that the Eskimo lives almost entirely on protein and fat. In times of plenty he may eat as much as four kilograms (nine pounds) of meat daily. He certainly gets a little carbo-hydrate from seaweed, the young shoots of angelica, mussels and from the glycogen contained in the skin of young whales, but all this yields little more than 8% of the calories of his food. Contrast this with the diet of a European in which carbo-hydrates form 67% of the calories of his food. It is true that the Eskimo eats nearly three times as much fat in his food as does the European, but what of the excessive amounts of protein that he consumes? It is broken down into amino acids—the building stones of the body as these fragments have been called. Some of these fragments produce large amounts of glucose, which provides a great part of the energy the Eskimo requires. But there is another source of glucose supply besides that which comes from carbo-hydrates and protein. It is well known that glycerol results from the breaking down of fats, that glycerol is capable of being entirely converted into glucose in phlorhizinized dogs and that it increases the glycosuria of the diabetic.

Woodyatt calculates that one hundred grammes of fat catabolized in the body may set free ten grammes of glycerol and so yield ten grammes of glucose. Thus we arrive at the conclusions that carbo-hydrates, proteins and fats all yield glucose, that the carbo-hydrates are completely converted into glucose and that 58% of the protein and 10% of the fat may be converted into glucose.

Acidosis.

It is common knowledge that acids accumulate in the blood and tissue fluids in various disorders of the body, *exempli gratia,* in nephritis, in diabetes, in starvation, in hyperemesis of pregnancy. In nephritis there is a failure on the part of the kidney to excrete those acids which are the products of normal metabolism. In diabetes, in starvation and in excessive and prolonged vomiting, which really is starvation, foreign acids are added to the blood. In all these conditions we may have the usual signs of acidosis because the excess of acids—normal or abnormal—produces the same effects, *id est,* the depletion of the stores of the bicarbonate of the blood, causing changes in the carbon dioxide of the alveolar air, in the reserve alkalinity of the body and in the acid excretion by the kidney. In diabetes and in starvation the acids which accumulate, are diacetic acid and β-oxybutyric acid. These acids are the result chiefly of a faulty metabolism of fat. For the complete combustion of fat in the animal body a certain amount of carbo-hydrate must be burned at the same time.

We have seen that fat is not so readily burned as sugar. It needs the fire of the burning sugar to burn it completely. If the carbo-hydrates are not burned in sufficient quantities, the fats are incompletely burned. Intermediate products or products of incomplete combustion result. These products are diacetic acid and β-oxybutyric acid, acetone or ketone bodies. Such incomplete combustion occurs in diabetes and in starvation. In diabetes because there is a disability to burn up sugar and in starvation because there is a deficiency of sugar to be burned. In both conditions, however, the production of these acid bodies resulting in acidosis is due to the same fundamental cause, namely, an improper or ill-balanced metabolism of fat and carbo-hydrate. But it must also be borne in mind that protein may yield these same ketone bodies, certainly not to the same amount, when there is a defective combustion of carbo-hydrates. In the case of a normal individual who abstains entirely from food, it is found that for the first few days none of these acid bodies appear in the urine. The stores of glycogen in the liver and muscles are sufficient for the complete combustion of fat. The proper relation between carbo-hydrate and fat combustion has been maintained. Later on these acid bodies appear, for the glycogen store has now been depleted and the fat cannot now be thoroughly burned. If the starvation is prolonged, the fat supplies as well as the carbo-hydrate supplies are used up, the proteins alone remain for the maintenance of life. During this period the acid bodies will diminish for the reason that in the metabolism of protein, as we have already mentioned, about one-half of the protein is converted into glucose which by combustion burns up or helps to burn up the other half.

For these reasons in very fat persons during starvation marked acidosis will result, but in very lean persons during starvation there will not be marked acidosis, because there is little fat to be burned. The organism must depend chiefly upon its protein for fuel and the protein, as we have seen, provides its own sugar during metabolism for its more or less complete combustion.

In diabetes the same principles apply. The power to use carbo-hydrates is wanting. There must be a balance between the burning of fats and carbo-hydrates and, as this balance is disturbed, the diabetic can only completely oxidize a smaller amount of fat.

To state the case briefly, an amount of fat which a healthy body can burn up, is imperfectly burned by the diabetic and acidosis results.

RANDOM OBSERVATIONS ON MITE INFESTATIONS OF MAN.

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THE infestation of the human host by various forms of mites is neither rare nor unusual and the species implicated are numerous. In spite, however, of the great contributions to our knowledge of insect pests in Australia which have been made by

J. Burton Cleland, T. Harvey Johnston, S. J. Johnston, Georgina Sweet, Ferguson, Breinl, Priestley, Nicoll and others, the closest scrutiny of the literature reveals only a few scattered observations regarding mite infestation in Australia.

Cases of skin irritation definitely traceable to *Sarcoptes hominis*, *Lælaps agilis* (parasitic upon *Mus decumanus*), *Leptus* species, *Dermanyssus avium* (the common fowl mite) and *Liponyssus bursa*, Berlese (the tropical fowl mite) have all been recorded and gamasids from hens, starlings, English sparrows (probably of one or the other of the last two types mentioned above) have been reported in several instances. In one instance the pest was such that it was necessary to remove the roof of a church and to clear out the nests of the birds which had established themselves amongst the rafters, before the congregation could assemble in comfort.

"Grocer's itch," "grain itch" and "copra itch" are other well-known varieties of skin irritation, the last being especially familiar to workers in New Guinea, while the so-called "red spider," *Leptus* (*Trombicula?*) species, also mentioned by Cleland, is found in many parts of Queensland and New Guinea and produces the maddening irritation known as "scrub-itch."

The attention of the writer was directed towards the question of mite infestation of the human host by the study of the affinities existing between the epidemic glandular fever of Queensland and Japanese river fever, the fever conveyed in Japan by the larval mite, *Microtrombidium* (*Trombicula?*) *akamushi*, which so closely resembles the local *Leptus* species.

The search amongst the literature resulting in a meagre return, attention was directed towards collections made from time to time at previous dates by Mr. Gerald F. Hill, the entomologist to the Institute of Tropical Medicine. It was found that mites of the most varying kinds and species parasitic on many varying hosts were extremely prevalent in the tropical localities of Australia and its dependencies.

Thus a large unidentified mite almost the size of a small cattle tick was discovered upon one of the *Paussidæ*. A small red tick was found on a specimen of *Tabanus avidus* from Magnetic Island, near Townsville, North Queensland. Three separate species (unidentified) were obtained from the bodies of queen termites collected in northern Australia. Specimens of *Mansonioides uniformis* from Bioto, in Papua, were found to be swarming with a variety of little white mite, one or more species were found in enormous numbers in cow-dung and besides the form which causes "red spider" itch or "scrub itch" (and possibly the "black soil itch" of Camooweal, West Queensland), numerous free-living species were found attacking tabanid larvæ in breeding jars.

By a curious coincidence, during the course of these observations the writer disturbed a pigeon's nest on the eaves immediately above a window of one of the laboratories at the Australian Institute of Tropical Medicine and was showered with specimens of the tropical fowl mite, *Liponyssus bursa*, while about the same time many specimens of the same mite engorged with blood were sent in from patients in Townsville by Dr. T. Gordon Ross. These cases are interesting as being the first reported from tropical Australia so far as can be ascertained and Dr. Ross has kindly attached the following histories:

CASE I.—Mr. A.B., aged forty-four years, stated that on getting up one morning he felt a slight itching around his ears and neck. On rubbing his hand over his neck he found a few small insects and then found a few more on his pillow. He did not feel anything further during the day, but next day on examining his pillow again he saw a number of insects and got a few on his face and shoulders. This occurred each morning for several days. There was no irritation or discomfort during the rest of the day. On examination no rash was visible on the patient's skin, no marks of insect bites, but some of the insects collected by the patient were found to contain blood. The patient used a weak "Lysol" lotion for sponging every morning and soaked his pillow slips in lotion when insects were discovered. After a few days no more were seen.

CASE II.—Mr. C.D., aged forty-five years, complained of a severe itching on the limbs and trunk which he had had for several weeks. On examination a rash was found which appeared to be identical with scabies, but there were not many pustules and the rash was not present between the fingers. He stated that he was in the habit of handling fowls a good deal and often on putting his hand into the nests would get his hand and forearm covered with little black insects. These, however, usually left his body very quickly and he was not apparently troubled by them when away from the fowls. He was asked to report again and bring some of the "fowl lice" for examination. Up to the present he has not done so.

Liponyssus bursa (Berlese) is widely distributed. It has been reported from the following localities among others: North and South Nigeria, Zanzibar, Nyassaland, Zululand. Conoro Island, Mauritius, India, China, United States of America, Bahamas, Trinidad, South America and Australia.

In Australia Cleland has reported instances from Cremorne, Sydney, New South Wales, and from Port Pirie, South Australia. The symptoms in these instances were those of intense irritation and rash.

Cases have also been reported by Howell from Achmednagar, India, where a lady suffered from a very bad irritation of the skin, alleged to be caused by the mite. It raised small red lumps with white tops. The appearance suggested that the insect burrowed and the irritation was intense even for days after the bite.

While it is not suggested that the mite in question or any other tropical Australian variety is necessarily the disseminator of any disease, the facts that it will attack man and that Hirst claims it to be the possible transmitter of spirochætosis in fowls render its presence in an area where there is endemic a disease closely resembling the mite-borne river fever of Japan, a matter of interest. Moreover, the very great prevalence of other mites of many varieties and the frequency of rashes and fevers of an unknown origin in some of the areas where they are prevalent, indicate that an investigation into the Australian tropical acarines is called for and might prove of importance.

Summary.

1. Investigation, initiated in the search for possible affinities between the epidemic glandular fever of Queensland and Japanese river fever, revealed

the presence in tropical Australia and its dependencies of a considerable number of varying species of mites.

2. During the course of this investigation two cases of infestation by *Liponyssus bursa* (Berlese) came to the notice of the writer. So far as can be learned, these cases which are here recorded are the first reported from these areas, though doubtless by no means the first seen.

3. The possibility of the presence of mite-borne diseases in tropical Australia suggests that a study of the local mites would be desirable and possibly important.

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Reports of Cases.

ANAPHYLAXIS WITH SUDDEN ONSET.

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THE following history is thought worthy of report on account of the severity of the symptoms and the rarity of the condition:

Clinical History.

J.S., male, *ætatis* nineteen years, was admitted to the out-patient department of the Ipswich Hospital at 8.30 p.m. on April 3, 1923. While playing football he had received a cut over the left eyebrow five centimetres (two inches) long.

On examination no head, abdominal or chest injury was detected. His wound was cleansed, sutured and dressed. He was given anti-tetanic serum (1,500 units) after having denied ever receiving previous serum treatment. After three-quarters of an hour and apparently feeling perfectly normal he left the hospital in the company of a friend.

At 10.15 p.m. (ten minutes after leaving the hospital) he was again brought in by a police constable, who supposed him to be intoxicated. A few minutes after leaving he had felt cold and ill and had commenced to vomit, first stomach contents and later watery fluid.

On examination his face was seen to be drawn and pallid, his pupils were dilated and his eyes rolled upwards. The skin was cold, moist and clammy. He was pulseless and the heart beats could be heard but faintly on auscultation. In fact, he was absolutely collapsed; his mind was clear and he could talk quite rationally, but he thought that he was going to die.

He was put to bed, was surrounded with hot water bottles and was given a hypodermic injection of 0.003 gramme (one-twentieth of a grain) of strychnine. The foot of the bed was raised and saline solution was administered continuously *per rectum*. His temperature was 35.3° C. (95.6° F.). His respirations numbered fourteen per minute. No head, chest or abdominal injury could be detected.

At 10.45 p.m. one cubic centimetre of pituitrin was given hypodermically and his skin became warmer. His eyelids were swollen and the vomiting continued. He complained greatly of thirst.

At 11.15 p.m. a second hypodermic injection of 0.003 gramme (one-twentieth of a grain) of strychnine was given.

At 12.30 a.m. he was seen by Dr. Trumpy and by this time was vomiting blood-stained fluid and a rash had developed on his limbs. At 1 a.m. 0.3 cubic centimetres of adrenalin (one in 1,000) were given and an hour later he was much improved. The temperature was 37.3° C. (99.7° F.), the pulse had improved in rate and volume, but was still uncountable.

At 2.15 a.m. one cubic centimetre of pituitrin was injected and from this stage he gradually improved.

At 8.30 a.m. on April 4, 1923, his rash had disappeared, vomiting had ceased, his temperature was 37.2° C. (98.6° F.), his pulse rate was 120 and his respirations numbered eighteen per minute. He had retained two and a quarter litres (four pints) of saline solution, which had been administered *per rectum*. He was given a fluid diet and made an uneventful recovery.

He was sitting up in bed on April 6, 1923, and on April 8, 1923, was taking a full diet. He was discharged on April 9, 1923.

At 4.30 p.m. on the same day, ten minutes after returning home, he began to develope pruritus of the limbs and body.

At 5.30 p.m. he was seen by Dr. Paterson, who found a well-marked rash present on the body and limbs, together with a swollen neck, stertorous breathing, cyanosis, œdema of the glottis and expectoration of a quantity of clear, frothy, colourless fluid.

He was re-admitted to hospital at 7.30 p.m. On examination the temperature was found to be normal, the pulse rate was 96 and the respirations numbered 24 per minute. Nothing abnormal was detected in the heart, abdomen or nervous system. Examination of the lungs showed moist sounds to be present all over the chest. A well-marked erythematous rash was present on the trunk and limbs, the eyelids were puffy, the throat was red and injected, the uvula was œdematous, while the neck was now no longer swollen and there was no difficulty in breathing. Sputum was now more scanty and the patient was feeling very ill. Calcium lactate was given every four hours in doses of one gramme (fifteen grains) and calamine lotion was applied externally.

At 10 p.m. he felt more comfortable, but complained of itching. A hypodermic injection of morphine of 0.01 gramme (one-sixth of a grain) was given.

On April 10, 1923, he complained of pain in the joints, especially the shoulder, knee and hip joints. These were swollen and contained fluid in slight amount. His recovery was rapid and uneventful. He was dicharged on April 15, 1923.

Comment.

1. The patient denied having had any previous serum treatment, but after much questioning admitted to having had three injections of anti-influenza serum in 1918.

2. He developed serum sickness seven days after his injection on April 3, 1923, and this rather confirms the diagnosis of anaphylactic shock on April 3, 1923.

3. The anaphylactic shock coming on three-quarters of an hour after the injection is interesting.

4. Kolman says: "Serum sickness and anaphylactic shock are more prone to follow anti-tetanic serum than anti-diphtheritic serum on account of the former being less concentrated and due to the less efficient removal of toxic bodies."

5. Only forty cases of anaphylactic shock are reported following within a few minutes of injection of serum and are characterized by dyspnœa, prostration and death. In one series of cases 0.9% of the patients showed signs of anaphylactic shock within twenty-four hours.