colouring action of acids, and we may safely say that our bacillus in almost every detail corresponds to the leprosy bacillus in the body; and coupling this with the fact that in two out of three cases examined we got identical results, and that the method used was one which held out most hope for success, we do not hesitate to consider our artificially cultivated bacillus to be the true bacillus of leprosy.

We tried first of all eggs, which were prepared and inoculated in the manner laid down in Hueppe's work *Die Methoden der Bakterienforschung*—a method by which Professor Max Wolff, of Berlin, succeeded in cultivating the actinomycetes. This gave us no result, as hardly any growth took place, most probably due to the deficiency of oxygen, for we had found that our bacillus is very aerobic. Acid gelatine and agar-agar likewise were of no avail.

On returning to our gelatine cultivations the following was observed. As described in our first communication, after inoculating a gelatine peptone tube the first thing noticed is the formation of a characteristic skin floating on the surface of the liquefied gelatine. The gelatine liquefies quite uniformly and slowly. The liquid gelatine under the pellicle is "stringy" and "mucoid." When the growth is two weeks old an opaque whitish sediment will be observed at the boundary line between the liquefied and non-liquefied gelatine. On removing material from this sediment it was found so mucoid as to be removed with difficulty. Heating the cover glasses carefully, and staining them according to Ziehl's method, we found that all bacilli retained the stain most beautifully. The same result was obtained with all the tubes examined, amounting to over twenty.

There can thus be no doubt that our bacillus also reacts like the leprosy bacillus, and in fact it only differs from it in this, that it takes up methyl-blue much more readily than the leprosy bacillus from tissues does. Staining the bacilli from the above sediment according to Gram's method, we have also obtained pictures showing the beaded appearance well, so that in this again our bacillus agrees with the leprosy bacillus.

A successful animal experiment would at once decide whether our claims are justified. So far, however, they continue to be in vain, and perhaps also but little result may be expected from them; for leprosy is so exclusively a human disease that one can hardly hope to produce the disease in animals. So far Melcher and Ortmann have been the only ones who succeeded in causing a dissemination of the leprosy bacillus after implanting a small nodule of leprosy tissue into the anterior chamber of a rabbit. We must therefore, for the present, depend on morphological and tinctorial characters. Now as far as these are concerned, we have brought also the last proof that our artificially cultivated bacillus is identical with the leprosy bacillus.

Several questions naturally arise in connection with the life-history of this bacillus. First, the changes in the staining reactions tend to decrease the value of such methods as are generally used in the diagnosis of the tubercle or leprosy bacillus. However, this is a point of only secondary importance, as other means exist which will enable the bacteriologist to differentiate a doubtful microbe.

The second question is, how are we to explain these changes in staining during the lifetime of one bacillus? As far as our bacillus is concerned, we can hardly doubt that the cause of the increased resistance against acids must be sought in the mucoid process initiated by the bacillus. On making careful preparations, it may be easily ascertained that many of the bacilli are enveloped in a capsule. This lends great support to Ehrlich's view with regard to the staining of the tubercle bacilli. The capsule would protect the bacillus from the decolorising action of the acids. In fact, we have found that, if we wish to bring out the shape of the bacilli properly, we must at this stage of their development decolorise more vigorously.

We have so far called the changes "mucoid." Examining the liquefied gelatine chemically, it has been found, as a matter of fact, to contain a considerable quantity of mucin body. This is all the more interesting, as, following the instructions of Hankin, we have succeeded in extracting a body from leper tissue, which, if not a mucin, is at least very closely allied to this class of organic compounds. On these points, however, we shall report at length at some future date. The

object of this paper was to state the reasons which led us to believe that the bacillus cultivated by us is in reality the leprosy bacillus.

AINHUM ON THE WEST COAST OF AFRICA.

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THE word "ainhum" signifies "to saw," and is applied by the Africans to a peculiar disease characterised by the formation of a constriction at the root of the small toe. The name of "sukha pakla," which means "dry suppuration," is applied to it by the Hindus. The disease has been observed in India among the Mahomedans and Hindus by Drs. Crombie and Wise; in Brazil among the negroes and creoles by Drs. Silva, Lima, and Seixas; and on the Gold Coast by Dr. Eyles among the Negroes.

The fifth toe is the one usually affected, but the disease has been once noticed in the fourth toe, and Dr. Eyles mentions a case in which the second finger was affected. It is mentioned as being symmetrical, but this is not usual, as out of twelve cases I have noted only two in which both feet were affected. The left foot seems to be more frequently attacked than the right, the disease occurring nine times out of twelve on this side. The race of negroes called Krumen who inhabit the coast about Cape Palmas, and who emigrate as workmen to the different stations along the coast from the Gambia to Fernando Po, seem to be particularly liable, as out of the twelve cases mentioned ten occurred in these people.

The disease has only been known to attack coloured races, and seems to occur solely in those who go about with their feet unprotected; it is worthy of note that the Krumen above mentioned have been wearing brass or copper rings on their fifth toes for generations. The nature of the soil inhabited by those affected may bear some relation to the disease, as the sandy districts of West Africa produce most of the cases. I have only noticed the disease in middle and advanced life, and it certainly occurs more frequently in men than in women, only three of the above cases occurring in the latter. Occupation I believe exerts a considerable influence in producing the disease; those men who are labourers and who are constantly employed carrying weights being most frequently affected. In fact, a combination of the above influences would materially assist in producing the constriction, irritation, and hypertrophy which characterise the disease. Dr. Seixas mentions that the disease occurs among those wearing boots as well as those who go barefooted; but I think this can be accounted for by the fact that natives who do wear boots or shoes get rid of them on every opportunity.

The disease begins by an extension in depth of the digito-plantar furrow, which at the same time extends round the sides of the toe, and ultimately joins on the dorsum, being accompanied by hypertrophy of the toe. Both these processes advance until nothing is left but a small fibrous pedicle attaching a ball of hypertrophied tissue to the foot. On section, the parts at the distal end of the toe may present a normal character, but on passing towards the furrow, the cuticle and true skin are seen to be hypertrophied, the fibrous tissue is increased, and eventually replaces the bone, which undergoes a process of rarefying osteitis.

Dr. Eyles has carefully investigated the histology of the disease, and accounts for the formation of the furrow as follows: there is a direct ingrowth of epithelium, with a corresponding depression of the surface due to very rapid hyperplasia, which pushes down and strangles the papillæ, thus cutting off the blood supply from the epithelial cells, and causing them to undergo a horny change, while the increased pressure sets up vascular changes which influence the hypertrophy of the more distant portion of the toe.

Accompanying the constriction is often observed a small ulcer in the bottom or side of the fold; it is not a constant phenomenon, and does not occur early in the disease, but arises from direct injury subsequent to the toe becoming unmanageable, and from dirt or sand lodging in the furrow. It is the pressure on this ulcer that gives rise to the great pain sometimes complained of.

Ainhum is a disease of some years' duration, and is seldom seen in hospital early in its history, as patients do not apply for treatment until inconvenienced by the toe; this inconvenience arises from the attenuated pedicle being unable to support the toe, which consequently bends when taken off the ground, and is caught under the foot in walking, occasioning considerable pain, particularly in those cases where the ulcer is present. The duration of the disease has been given at from one to fifteen years; the former time is probably much too short. The disease causes no constitutional disturbance, and is looked on by the natives as such a slight ailment, that they will themselves perform amputation if the toe troubles them. When total spontaneous amputation occurs, the small wound left heals rapidly and naturally, leaving no remarkable appearance in the cicatrix.

RESECTION OF CARCINOMATOUS BOWEL PER RECTUM.

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Mrs. A., aged 44, was sent to me by Dr. Goodworth, of Winterton. For four days she had had acute abdominal pain, tympanites, vomiting, constipation, and tenesmus, with escape of blood and mucus. Two inches inside the anus the apex of an intussusception was felt, with a mass of epithelioma involving two-thirds of the circumference of the bowel, and extending upwards for nearly 2 inches. She was sent into the Hull Royal Infirmary, and after consultation with my colleagues I decided to remove the growth and reduce the intussusception. Previous to the operation there was hardness of the muscles over the left iliac fossa and a quick thready pulse.

Under ether the intussusception was found to be about 5 inches long. The long tube was passed into the lumen for about 6 inches without finding any obstruction. The mass of bowel and growth was readily extracted from the anus, and the left forefinger was passed into the lumen of the intussusception. With a curved needle nine silk sutures were passed through the two layers of bowel wall from the outer mucous lining into the lumen, then to the outside again in the direction of the circumference, each suture overlapping the previous one for $\frac{1}{4}$ inch. A ring was thus formed, causing firm apposition of the serous surfaces 1 inch above the margin of the growth. The mass was then removed $\frac{3}{4}$ inch below the ring of sutures. On being returned into the rectum the intussusception rapidly receded out of reach.

Twenty-six hours after the operation, as the symptoms had not been relieved and no flatus or motion had been passed, ether was again given, and the abdomen opened over the sigmoid flexure. Gas escaped on opening the peritoneum, and pus welled up from the iliac fossa. The small intestines were distended, matted together, and firmly adherent over the sigmoid flexure, which could not be exposed. The wound was irrigated with boric acid solution, a Keith's tube put into the iliac fossa, and an artificial anus was made in the most prominent coil of the small intestine. The patient did not rally, but died in twelve hours.

Post-mortem Examination.—At the site of the resection the peritoneal surfaces were in close contact and there was no sign of inflammation. The bowel above was plugged with solid faeces for 6 inches; there was a collection of pus in the left iliac fossa, and the adjacent bowel was covered with tough lymph. The small intestines were firmly matted. No perforation was discoverable in the region of the sigmoid flexure. The artificial anus was found to be about the middle of the small intestine.

REMARKS.—The toughness of the adhesions and the amount of pus showed that the peritonitis was of at least three days' duration. The absence of inflammation at the site of resection negatives the idea of extravasation at the time of operation. If the peritonitis had not previously existed, I see no reason why this method of operating should not have been successful.

REPORTS TO THE SCIENTIFIC GRANTS COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION.

REPORT ON THE ACTION OF PANCREATIC AND RENNET EXTRACTS ON CASEIN.

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It has been a matter of occasional observation that milk will undergo clotting under the influence of pancreatic extracts. Besides this change, visible to the naked eye, Roberts has pointed out that though no actual clotting may occur, yet the casein in milk may undergo such an alteration by the action of pancreatic extracts that boiling will cause a curdling of the milk. Roberts applied to the casein when it had undergone this change the term metacasein. The experiments I have made indicate more clearly, perhaps, what this change is.

The existence of the metacasein was shown by Roberts to be merely transient. After the digestion of the milk with pancreatic extracts had lasted for a certain time, boiling a sample of the digesting mixture resulted in the formation of a few flocculi of heat-coagulated material. Later, this formation of coagulum became more marked, and after reaching a maximum gradually lessened till, finally, boiling produced no apparent result.

My earlier experiments were directed at ascertaining whether this metacasein was to be regarded as simple casein precipitated by the combined effect of heat and free fatty acid formed by the fat-splitting ferment of pancreatic extracts. I was able, however, to obtain specimens of pancreatic extracts which had lost their ptyolytic properties (the ferment determining these properties being, as pointed out by Grützner, by no means durable), and milk when treated with this form of extract caused the characteristic change to occur. I was able to ascertain that neither before nor after boiling was there any alteration of reaction in milk, alkaline at the commencement of the experiment. Since, moreover, pancreatic extracts lost their power if subjected to heat, it appears that the result was due to some ferment of the pancreas. Certain influences affect this change in the casein, most important being the presence of neutral salts. A small quantity of sodium chloride added to milk will considerably hasten the appearance of the metacasein reaction. Free acids and alkalis appear to have very much the same effect upon the ferment determining this change as upon the proteolytic ferment.

I next attempted to ascertain what relation there was between this metacasein-forming ferment and the pancreatic rennet ferment that has been previously described. It was first necessary to obtain some more exact information upon this latter ferment. Not every specimen of pancreatic extract will produce clotting of milk, and it was first necessary to see what circumstances determined the action. I found first that, taking a specimen of pancreatic extract which caused clotting when added in definite proportion, if this were added in too large an amount, no clotting occurred. Such clots as do occur become later perfectly liquid. If the milk be but slightly diluted, the probabilities of a clot occurring are much decreased. It has been pointed out that brine extracts are very much more potent in causing a clotting than are most other forms. This is simply the effect of the concomitant addition, with the extract, of neutral salt. If to specimens of milk, not clotting by the action of extract, sodium chloride be added to bring the total percentage of the liquid up to 5 per cent., clotting will occur under the influence of the extract, unless this be extremely weak. This result cannot be explained by the supposition that really the proteolytic ferment is being retarded, and therefore the rennet ferment has freer scope, since up to this percentage sodium chloride has an accelerating power upon the proteolytic action of pan-