



The CENTRAL AFRICAN JOURNAL OF MEDICINE

Dr. DAVID LIVINGSTONE

Vol. 5, No. 8.

CONTENTS

August, 1959.

ORIGINAL ARTICLES

Acute Eosinophilic Pneumonia Possibly due to Infection with <i>Ascaris Lumbricoides</i> Tropical Eosinophilic Syndrome	<i>I. M. Patz</i>	399
Mushroom Poisoning in Rhodesia	<i>Michael Gelfand and Hilliard Bernberg</i>	405
Pseudohypertrophic Muscular Dystrophy in Two Members of an African Family	<i>G. R. B. McCarter</i>	412
African Housing in Southern Rhodesia	<i>Frances Christie</i>	414
Medical Ethics	<i>M. I. Hirsch</i>	416
	<i>Maurice Davidson</i>	424

EDITORIALS

The Rhodesian Bee		429
Discussion on Cervical Spondylosis		430
Dr. Haydn Jones Morris, O.B.E., Retires		431
The Queen's Birthday Honours		432
Presentation of a Photographic Portrait of Her Royal Highness the Princess Margaret		433
Miss Barbara Rhoades, S.R.N.	The Royal Society	445
Retirement of Mr. J. H. G. Robertson	Book Reviews	447
The Salisbury Clinical Club	The Journal Library	449
Medical Education in East Africa	In the Federal Assembly	450
The Gwelo Memorial Cottages, Gwelo	The Ulcer that Never Was	453
At the Harare Hospital Staff Round	Latest Pharmaceutical Preparations	455

PUBLISHED MONTHLY, ANNUAL SUBSCRIPTION £2 2s. 0d.

Registered at the General Post Office as a Newspaper.

The Central African Journal of Medicine

Volume 5

AUGUST, 1959

No. 8

Acute Eosinophilic Pneumonia Possibly due to Infection with *Ascaris Lumbricoides*

BY

I. M. PATZ, M.B., B.CH. (Rand)
Middelburg, Transvaal, South Africa.

During a 48-hour period in November, 1956, 12 adult Bantu presented with acute pulmonary symptoms of four to six days' duration. Eleven of the patients worked and lived at a brickfield about one mile from the town of Middelburg, Transvaal. The patient who did not work at the brickfield, but in the town itself, had his home at the brickfield. It was the close association of these patients which suggested a common cause for the illness.

Nine of the patients were admitted to hospital for investigation; the remaining three refused hospital admission. Three of the cases had been seen in the previous week with a mild urticarial rash, the significance of which was not appreciated at the time.

The place of work was carefully examined for possible causes of the illness, such as chemicals and dust, but none could be incriminated. The only common factor was the story that approximately 12 days previously they had all attended a party and beer drink illicitly held at the brickfield.

The method of preparation of the food and drink consumed at the party or even the exact locality of the party could not be discovered, as none of the patients was prepared to divulge any information on the subject.

CLINICAL FEATURES

The symptoms were as follows: About four to six days prior to presenting themselves, the patients developed malaise, headache and generalised body pains. This was soon followed

by rigors, pyrexia and a dry hacking cough. The symptoms were at their height at the time of the first consultation. The cough was almost continuous, exhausting and very much worse at night. Vomiting was a common accompaniment of the cough. The sputum was scanty and white in colour; only one case showed small streaks of blood in the sputum. Wheezing was a prominent feature in all the cases.

On examination they were all pyrexial, the temperature varying from 100° F. to 104° F. The pulse was not correspondingly raised in all the cases. They were all extremely breathless and coughed continuously during the examination. On auscultation, all the cases showed diffuse rhonchi and fine crepitations, but no clinical signs of consolidation were detected. At night the wheezing was very prominent and it sounded as if a group of asthmatics had been placed in one ward. For the next four to five days the patients were very ill, after which resolution of the symptoms and signs was rapid. All the patients were discharged by the tenth day and were back at work a fortnight after admission to hospital. There were no relapses and the patients have remained well since discharge.

RADIOLOGICAL FEATURES

Radiological features varied from increased lung markings to a diffuse bronchopneumonic mottling involving both lung fields, including the apices. The symptoms and signs did not necessarily correspond with the radiological findings. Some of the milder clinical cases had severe pulmonary involvement radiologically and *vice versa*. Repeat radiographs done one week later showed radiological improvement, but not to the same degree as one would have expected from the rapid clinical improvement. The radiographs done seven to eight months later on nine cases that were traced showed complete resolution. More frequent radiological examinations were not possible.

LABORATORY INVESTIGATIONS

The further investigations included repeated blood counts, sputum examinations, stool and urine examinations for parasites, virus studies on stools and sputa, bilharzial complement fixation tests and histoplasmin skin tests.

Analysis of the results was as follows: All the cases showed an eosinophilia varying from 7 per cent. to 63.5 per cent., with an average level between 20 per cent. and 40 per cent. All the sputa examined showed a great predominance of eosinophils. The sputa examined did not contain mites or helminth larvae. However, only single specimens of sputum were examined and 24-hour specimens were not obtained for examination. Repeat blood counts four weeks and eight weeks later showed that the eosinophilia had cleared in almost all the cases, one or two cases still showing a slight elevation in the percentage of eosinophils. Three of the cases gave a positive history of bilharzial infection previously. They all came from endemic areas. Two of them showed ova of *S. haematobium* in the urine at the onset of the illness and the third case had ova of *S. mansoni* in the stools. This case had previously been diagnosed at another hospital. None of the other cases had bilharzial ova at the onset of the illness or

showed their presence in urine or stool specimens, repeatedly examined over the next four months.

The results of the bilharzial complement fixation tests were very variable. The test varied from positive to doubtful and negative in the cases passing ova and became positive in one case previously negative and in whom no ova could be found in repeated urine and stool examinations.

Of the twelve cases seen, two admitted to the passing of round worms in the stools previously, but eight of the stools examined at the onset of the illness showed the presence of ova of *Ascaris lumbricoides*. Of the four negative cases, three had ova in the stools eight to 16 weeks later. One patient (F.M.) had shown no ova in his stool after a period of seven months. This patient, incidentally, had the lowest eosinophil count (7 per cent.), but had extensive pulmonary involvement radiologically. The follow-up in this case was unsatisfactory, as he left his place of employment to return to his home and would return for further investigation only at irregular intervals. It is interesting that several months later most of the cases presented themselves voluntarily for treatment, as they were passing large numbers of round worms in their stools

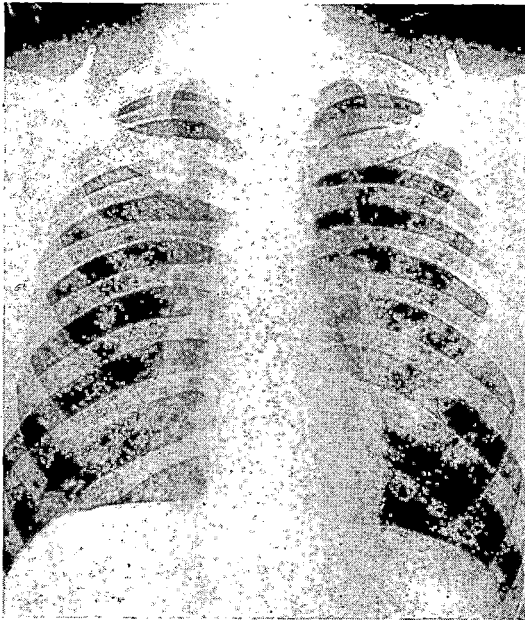


Fig. 1 (a)—22/11/56. The lung fields show mottling throughout, with emphasis on the left mid zone.

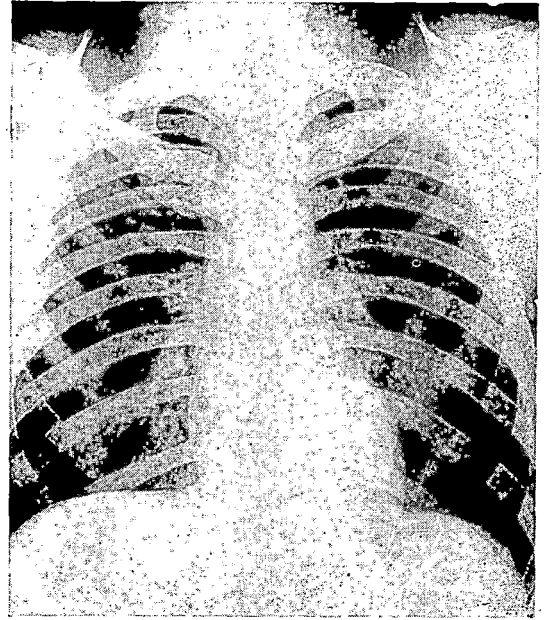


Fig. 1 (b)—12/6/57—Complete clearing has now occurred.

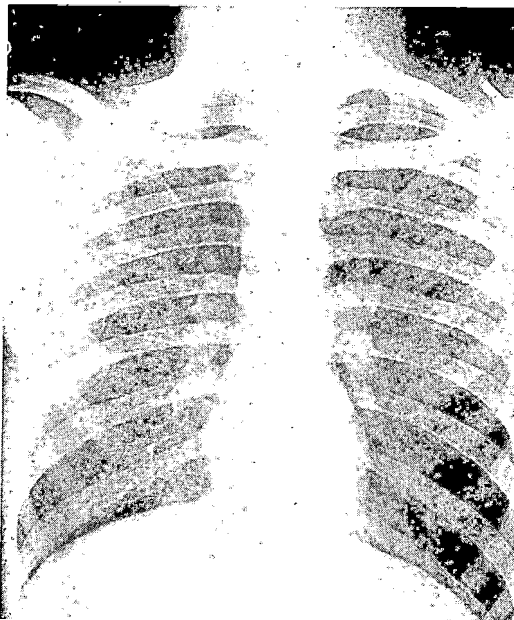


Fig. 2 (a)—22/11/56. The lung fields display fine mottling throughout.

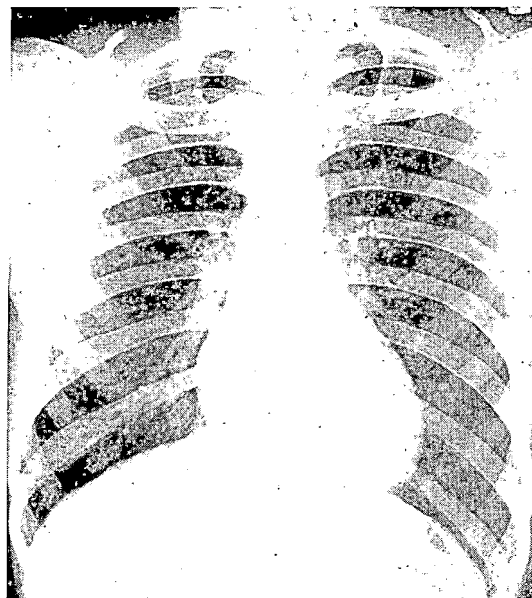


Fig. 2 (b)—12/6/57. There is still some increase in basal lung narrowing. A bronchiectatic basis is probably responsible for these changes. The rest of the lung fields are clear.

and some were vomiting worms as well. One of the cases (J.S.) was operated on about six months later for an acute appendicitis, and large numbers of round worms could be felt in the small bowel.

On discovering the eosinophilia in these patients, ten non-affected workers at the brickworks were screened and two were found to have eosinophil counts of 37 per cent. and 28 per cent. respectively. Neither of these had any symptoms. One had ascaris ova in his stools at the original examination, while the second case, having no ova in the stools at the first examination, was passing ova in his stools eight weeks later. Unfortunately they were not X-rayed to see whether they had asymptomatic pulmonary infiltrations. Another brickfield worker, but not one of those examined previously, was seen several months later with symptoms and signs of a subacute intestinal obstruction. On the grounds of his coming from an area known to be heavily infested with *A. lumbricoides*, he was given an appropriate anthelmintic with resultant passage of large numbers of round worms and cure of his illness.

CASE HISTORIES

Case 1.—M.B., aged 25 years, was seen on 21st November, 1956. He gave a history of generalised

body pains and headache commencing six days previously. Cough began the following day, as well as rigors, pyrexia and severe headache. The cough was exhausting and productive of only small quantities of white sputum. On examination, he appeared to be very ill. Temperature 103.4° F., respiration 40 per minute, pulse rate 120 per minute. Diffuse rhonchi and fine crepitations were heard throughout the whole of both lung fields. The radiological findings are demonstrated in Fig. 1. On 23rd November, 1956, he was still very ill and there was very little change in the signs and symptoms. By 27th November, 1956, he was better, but still troubled by the cough. On 30th November, 1956, he felt quite well and there were no physical signs. On 26th November, 1956, his blood count showed 63.5 per cent. eosinophils out of a total of 24,000 white blood cells. On 17th January, 1957, the eosinophils numbered 0.5 per cent. of his total white blood cell count.

Ascaris ova were present in the stools at the onset of the illness.

Case 2.—J.M., aged 28 years, was also seen on 21st November, 1956. He had been ill for five days. He developed sudden onset of pyrexia and rigors, accompanied by generalised body pains and headache. The next day he was coughing. The cough was almost continuous and productive of only small quantities of white sputum. On examination, the temperature was 102° F., respiration rate 30 per minute and pulse rate 100 per minute. Rhonchi and fine crepitations were heard throughout both lung fields. The patient's condition improved over the next few days and he was recovered by the ninth day after admission to hospital. The eosinophil count dropped from 16.5 per cent. of 8,000 leucocytes at the time of the illness to 4 per

cent. of 10,000 two months later. This patient had no ascaris ova in his stools on 28th November, 1956, but ova were detected after repeated examinations on 20th March, 1957. The radiological features are demonstrated in Fig. 2.

Case 3.—J.S., aged 17 years, was seen on 23rd November, 1956, with a three-day history of headache, backache, cough, dyspnoea, pyrexia and rigors. On examination, he did not appear to be as ill as the other cases. Temperature 100° F., respiration rate 26 per minute and pulse rate 100 per minute. There were rhonchi and crepitations throughout both lung

again on the 21st November, 1956, complaining now of generalised body pains, headache, rigors and pyrexia for the past six days. He had been coughing for the last five days and was short of breath for one day. On examination, he was very distressed. The temperature was 101° F., pulse rate 110 per minute and respiration rate 30 per minute. He was sweating profusely. Rhonchi and crepitations were heard throughout both lung fields. The next day (22nd November, 1956) there was slight blood streaking of the sputum. Symptoms and signs gradually cleared, and by 30th November, 1956, he felt quite well, but there were still rhonchi and crepitations present on auscultation. On 22nd November, 1956, the eosinophils numbered 7 per cent. of 9,100 white blood cells. The sputum, however, contained numerous eosinophils. This patient was very difficult to follow up, but stool examinations remained negative when seen six months later.

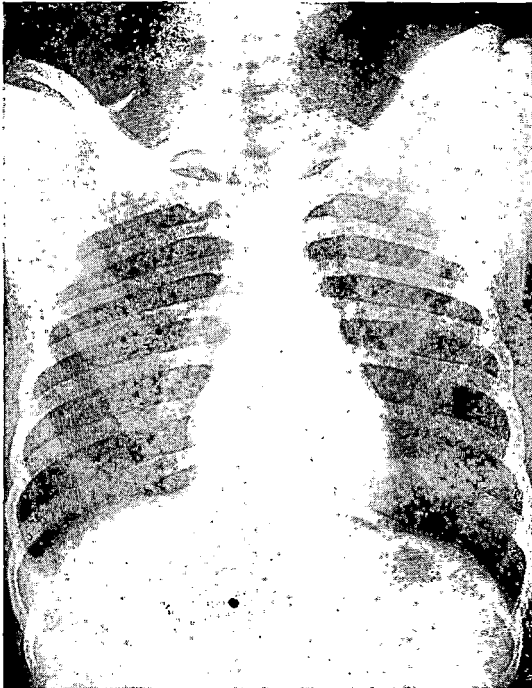


Fig. 3 (a)—24/11/56. Coarse mottled shadowing is present in both upper zones. The left lower zone also shows mottling, but in a lesser degree.



Fig. 3 (b)—5/6/57. The lung fields are within normal limits.

fields, the signs being far more extensive than his clinical illness suggested. This patient recovered rapidly and by the seventh day was quite well. On 26th November, 1956, his blood count showed an eosinophilia of 23.3 per cent. out of a total of 11,500 white blood cells. On 21st January, 1957, there was a 3.5 per cent. eosinophilia out of a total of 10,100 white blood cells. At onset of illness there were no ascaris ova in the stools (24th November, 1956). On 26th January, 1957, ova of *A. lumbricoides* were found in the stools. The radiological findings are described in Fig. 3. When operated on for acute appendicitis six months later, numerous round worms could be felt in the small bowel.

Case 4.—F.M., aged 23 years, was seen on 12th November, 1956, complaining of a generalised urticarial eruption (one of the three cases presenting with an urticarial rash prior to the onset of the major illness). He was given an antihistaminic drug and was seen

DISCUSSION

Besides various parasitic and allergic conditions causing pulmonary infiltration, eosinophilia has been reported in many lung conditions. It is not intended to discuss the causes of pulmonary infiltrations associated with eosinophilia, but to limit the discussion to various features associated with this particular outbreak. Tropical eosinophilia as described by Weingarten (1943), Frimodt-Möller and Barton (1940), Ball (1950) and many others differs from the outbreak described in this paper, in that the cases described here had a short sharp illness without the sequelae and recurrences which are characteristic of tropical eosinophilia. The other unique

features were the epidemic nature of this outbreak and its localisation to one small area, pointing to a common agent as the cause.

Eleven of the 12 cases were ultimately excreting ascaris ova in the stools. Eight cases had ova at the commencement of the illness, and this is not surprising, as infection with ascaris is extremely common, especially in the Bantu population. It is not unreasonable that these cases had a super-added infection causing the pulmonary infiltration. If it is assumed that infection occurred at the week-end party held 12 days prior to the cases presenting themselves at the height of the illness, the sequence of events is very similar to that described by Koino (1922), who infected himself by swallowing 2,000 ova of *A. lumbricoides*. On the sixth day after the ingestion of the ova, chills, headache and cough occurred, and five to six days later, i.e., about the twelfth day, after swallowing the ova, the illness was at its height and characterised by continuous coughing, dyspnoea and wheezing. By the tenth day after onset the illness was subsiding. Most larvae were recovered from the sputum on the fifth day of the illness, viz., 178, and these disappeared rapidly from the sputum, so that by the tenth day of the illness no larvae were recovered from the sputum. The maximum recovery of larvae from the sputum coincided with the height of the illness. It will be seen that our patients became ill about the fifth or sixth day after the party, and the illness was at its height on about the twelfth day. It was only two to three days later, however, that the sputa were examined for parasites, as at an earlier stage the significance of examining the sputa for helminth larvae had not been appreciated. The sputa from the patients were therefore examined for larvae after the height of the illness had passed. This, together with the fact that only single specimens and not 24-hour specimens were examined, and the distance of the laboratory from the hospital (100 miles), militated against the finding of larvae in the sputa.

The life cycle of *Ascaris lumbricoides* involves passage of the larvae through the lungs in all cases. It is therefore rather surprising that the pulmonary manifestations are not diagnosed more often clinically, as ascaris infection is one of the most universal of helminth infections. This is probably because symptoms in most cases are so slight as to be regarded as due to "upper respiratory infections," especially in children or infants who are most likely to become infected. Keller *et al.* (1932) have shown that there was

a significant increase in size of hilar shadows and density of bronchovascular marking in children harbouring *Ascaris lumbricoides* as compared with a control group having no such infection.

In endemic areas the population is being constantly re-infected, and this can occasionally be shown by finding worms in different stages of development in the same person (Yokogawa *et al.*, 1956). More often, however, an established intestinal infection apparently interferes with the development of young worms which enter the intestine subsequently, but does not prevent their migration to the liver and lungs, where in these instances there are relatively severe tissue reactions. This has been noted by Jung (1954) in his study of single-brood infections in children. The occasional ingestion of a few infective eggs by individuals with intestinal infections may cause only mild, if any, symptoms (Keller *et al.*, 1932). However, that it is not necessary to have heavy inoculations either in the presence or absence of existing intestinal infections to produce severe pulmonary infiltration has been shown by Vogel and Minning (1942), who found that as few larvae as 45 or less may produce frank illness and severe pulmonary changes. On the other hand, symptoms must generally be relative to the magnitude of the inoculum. Death apparently due to pulmonary larval ascariasis in an adult was recently reported by Beaver and Danaraj (1958). In this case numerous larvae were demonstrated in the bronchioles and the onset of the illness and the symptoms, though more severe, were essentially the same as were observed in the present series of cases. Experimentally, it has been shown that re-exposure to infection with the larval stages of certain helminths may lead to severe reactions. *Ascaris* larvae are retarded in their development and migration in previously exposed mice (Sprent and Chen, 1949). Necator larvae cause progressively greater skin reactions after repeated exposures to the human skin (Beaver, 1956), and the cercariae of schistosomes of birds similarly elicit greater response from re-exposed skin of man and other mammals as in swimmers' itch (de Meillon and Stoffberg, 1954; Olivier and Weinstein, 1953). At least some of the cases described in this paper must have resulted from heavy infections, as is suggested by the passage of large numbers of worms a few months later. It is the impression that those individuals who had previously been infected had a severer illness and they certainly showed the highest eosinophilia. The lowest eosinophilia of the series was found in

Case 4 (F.M.) and the mildest illness was seen in Case 3 (J.S.). In both of these cases no ova were found in the stools at the onset of illness. The other two patients who were not infected at the time of the illness showed lower eosinophil counts (16.5 per cent. and 13.5 per cent.) than most of those who were already infected. Eosinophil counts of more than 40 per cent. of the total white blood count were found only in individuals who harboured mature worms.

The evidence favouring *A. lumbricoides* infection as the cause of these cases of eosinophilia with pulmonary infiltration is mainly circumstantial, but it appears to the author as the most likely cause of the outbreak.

SUMMARY

An outbreak involving 12 cases of an acute illness associated with blood eosinophilia, asthmatic-like symptoms and the presence of radiologically evident pulmonary infiltrations is described. It is suggested that infection with *A. lumbricoides* was the cause of the condition.

REFERENCES

1. BALL, J. D. (1950). *Trans. Roy. Soc. trop. Med. and Hyg.*, 44, 237.
2. BEAVER, P. C. (1956). *Exper. Parasitol.*, 5, 587.
3. BEAVER, P. C. & DANARAJ, T. J. (1958). *Am. J. trop. Med. & Hyg.*, 7, 100.
4. DE MELLON, B. & STOFFBERG, N. (1954). *S. Afr. med. J.*, 28, 1062.
5. FRIMODT-MOLLER, C. & BARTON, R. M. (1940). *Indian med. Gaz.*, 75, 607.
6. JUNG, R. C. (1954). *J. Parasitol.*, 40, 405.
7. KELLER, A. E., HILLSTROM, H. T. & GASS, R. A. (1932). *J. Amer. med. Assoc.*, 99, 1249.
8. KOINO, S. (1922). *Jap. Med. World*, 2, 317.

9. OLIVIER, L. & WEINSTEIN, P. (1953). *J. Parasitol.*, 39, 280.
10. SPRENT, J. F. A. & CHEN, H. H. (1949). *J. infect. Dis.*, 84, 111.
11. VOGEL, H. & MINNING, W. (1942). *Beitr. Klin. Tuberk.*, 98, 620.
12. WEINGARTEN, R. J. (1943). *Lancet*, 1, 103.
13. YOKOGAWA, M. T., OSHIMA, S., SANO, M., KIHATA, S. SATO & KOMIYA, Y. (1956). *Bull. Inst. pub. Health*, 5, 2 (personal communication, P. C. Beaver).

Acknowledgments

I wish to thank the Superintendent of the Middelburg Hospital, Transvaal, for permission to publish this paper. I am indebted to all the persons who helped in the investigation and discussion of the cases. These include Dr. M. Gelfand, of Salisbury, S. Rhodesia; Drs. A. Wolpowitz and J. L. van Rhyn, radiologists to Middelburg Hospital; and Dr. M. Berk, of the Department of Radiology, Johannesburg Hospital.

All the investigations and photographs were done by the South African Institute of Medical Research through the good offices of Prof. J. F. Murray. His constant encouragement, interest and advice from the very beginning of this investigation made this paper possible.

My sincerest thanks are due to Prof. P. C. Beaver, of Tulane University, New Orleans, U.S.A., for the numerous suggestions he made during the preparation of the paper and for providing me with many of the references and reprints of articles, several of which were unobtainable in this country. He and his colleague, Dr. R. Jung, kindly corrected and made several useful additions to the final manuscript.

My thanks, too, to the typists, Mrs. P. Friedman and Mrs. R. Murning.



This work is licensed under a
Creative Commons
Attribution – NonCommercial - NoDerivs 3.0 License.

To view a copy of the license please see:
<http://creativecommons.org/licenses/by-nc-nd/3.0/>

This is a download from the BLDS Digital Library on OpenDocs
<http://opendocs.ids.ac.uk/opendocs/>