# EFFECTS OF *TRYPANOSOMA VIVAX* INFECTION ON FEMALE REPRODUCTION IN CATTLE

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# D E C L A R A T I O N

I hereby declare that this thesis has been written by me and that it is a record of my own research work. It has not been presented in any previous application for a higher degree. All quotations are specifically acknowledged by means of references.

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# D E D I C A T I O N

This thesis is dedicated to my children, IKECHUKU and NKECHI and those yet unborn. It is also dedicated to all my friends who stood by me during one of my most difficult periods.

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ABSTRACT

A group of 24, 3-year-old brucellosis free Zebu heifers obtained from the tse-tse fly free areas of Northern Nigeria were used to study the effects of Trypanosoma vivax, strain Y58, on: fertility, first, second and third trimester pregnancies and the effects of the reproductive status in heifers on the immunoglobulin M and G levels in bovine trypanosomiasis.

On the effects of T. vivax infection on bovine fertility, a group of 6 heifers from above was used. Four heifers in the follicular phase of estrus were inoculated with approximately $1.5 \times 10^6$ T. vivax while 2 others in the same phase were uninfected controls. All were bred by a proven bull. All the infected heifers developed fluctuating hyperthermia from the 5th day post infection rising on occasions to $40.3^{\circ}C$. Other clinical signs shown included, anemia, emaciation and lethargy. Rectal examination of the heifers 40 days post breeding showed that the 2 control heifers were pregnant while the 4 infected heifers were open. These infected heifers subsequently became anestrus for over 5 months. Different theories propagated to account for the infertility and anestrus included hyperthermia, release of trypanosomal neuraminidase and possible hypophysial lesion resulting in reproductive

hormonal imbalance.

On the effects of T. vivax infection on pregnancy, 18 of the heifers when pregnant were divided into 3 groups of 6 each. In the first group, 4 heifers were infected with T. vivax on day 60 (first trimester) of pregnancy while 2 others were uninfected controls. The second and third groups were similarly infected in the 4th (second trimester) and 7th (third trimester) months of pregnancy.

One infected heifer in the first trimester group aborted 39 days post infection while the remaining 3 had relatively normal gestation and parturition. In the second trimester group, the pregnancies were carried to term with normal deliveries. In the third trimester group, 3 infected heifers (75%) had premature deliveries while the 4th died about 24 hours after the full term calf was pulled out. All the control heifers had normal gestation and parturition.

No gross abnormalities were seen in the placentae of the infected heifers but histological sections of the infected third trimester pregnancy heifers revealed more mononuclear cells than in those of the uninfected controls. Postmortem examination of the premature calves showed lymphoadenopathy, splenomegally, serous atrophy of perirenal and pelvic

fat, epicardial petechial hemorrhages and blood-tinged peritoneal and pericardial fluids. Histologically there were slight myocardial hemorrhages and edema.

T. vivax was recovered in the blood of one of the premature calves. The calves of infected dams had lower birth-weights, and PCV than the calves of the control heifers. This work therefore demonstrates transplacental transmission of T. vivax in heifers.

Amongst the infected heifers in the 3 stages of pregnancy, clinical manifestations of the infection varied with heifers in the third trimester pregnancy showing more severe signs than those in the first and second trimester. There was also group variations in rectal temperature and PCV of the infected heifers.

Weekly serum samples collected from the non-pregnant, first, second and third trimester pregnant groups were quantitatively analysed for immunoglobulins M and G over a 15-week period. Kits were obtained from Miles Laboratories, England.

All the infected heifers showed significant rise in IgM levels when compared to the controls. 7 to 8 fold increases were recorded. There were however, differencies in the rate of increase and ability to maintain peak level responses (P 0.01) between the groups. Infected pregnant heifers in the first and second trimesters responded better than infected non-pregnant and infected heifers in the third trimester of pregnancy. In all the groups, the differences between the infected and the uninfected control heifers were highly significant (P 0.001).

The IgG results showed similar though gradual pattern of increases in the infected heifers. In the different groups, 1.2 to 2.5 - fold increases were recorded. These were significantly different from the levels in the uninfected control heifers (P 0.01). Again, pregnant heifers in the first and second trimesters responded better than non-pregnant and the third trimester pregnant heifers.

The results correlated with the observation that given the same conditions, clinical manifestations of T. vivax infection is more severe in non-pregnant than in pregnant heifers except those with advanced pregnancy. It is suggested that in using elevated IgM levels as a screening test for the diagnosis of T. vivax infection, consideration should be given to

the reproductive status of the animal in the interpretation of the test results.

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# I N T R O D U C T I O N

There are no accurate figures on the cattle population in Nigeria. Estimates have for years, been based on the returns from cattle tax "jangali" which is grossly inaccurate because cattle raisers tend to declare fewer animals for taxation than they actually possess. Similarly, data collected from vaccination campaigns are unreliable. This state of affairs has arisen because over 90% of the cattle in this country are still in the hands of the nomadic Fulanis (Nuru, 1974). However based on estimates from the Federal Livestock Department and the Food and Agricultural Organisation (FAO), the Green Revolution Report (1981) has accepted the figure of 9.3 million heads as a reasonable estimate of Nigeria's cattle population in 1981.

This cattle population is dominated by the Zebu breeds made up as follows: Bunaji, 51%; Rahaji, 14%; Sokoto Gudali, 11.5% Adamawa Gudali 11.5% and others 12.0%. In addition, various exotic breeds, whose suitability to our particular needs are still doubtful, have been introduced mainly for cross breeding with our indigenous breeds. They include the Holstein-Friesian, the South Devon, Brown Swiss, Jersey etc. It must be stated here that recent attention has been focused on the trypanotolerant breeds - the N'dama, Muturu and Keteku which together have a population of about 0.7 million (Green Revolution - Interim Report, 1981) and whose

potentials have not been fully investigated. It is expected that their trypanotolerant qualities would be exploited and the animals used for large scale beef production in the tse-tse infected areas of the South.

It has been estimated that 94% of the cattle population in Nigeria are located in the Northern States (FAO, 1966). The distribution is such that the traditional breeding areas are located in the drier Sahel and Sudan vegetation zones (Maclennan, 1970). These vegetation zones are arid for most of the year while the vast Guinea Savannah zone with its abundant pastures and water are not fully utilised for cattle production. This anomaly has been created due to tse-tse fly and trypanosomiasis. Thus, it can be said that the cattle population in this country is located in an area that is not the most ecologically suitable. According to the Green Revolution Report (1981), 600,000 to 700,000 $Km^2$ or 75% of the entire country is infested with tse-tse fly. This area, by extrapolation from the figures of Jasiorowski (1972) is potentially capable of supporting extra 1.2 million cattle. Attempts by the Federal Government to eradicate tse-tse flies in the areas otherwise suitable for cattle production (because of the availability of quality feed and water) have so far proved exhorbitant. This has cost well over N49.5 million in land and helicopter application of insecticides (Putt et al., 1980).

It has been remarked that the per capita meat consumption in Nigeria is very low (Osori, 1976). With the present rate of population growth, the problem is not likely to be solved soon even with the Green Revolution programme. According to the interim report of the study conducted by the World Bank and Nigerian Consultants for the Federal Ministry of Agriculture and the National Committee on Green Revolution (1981), Nigeria cannot be self sufficient in beef supply till 1986 and this will be possible only if all the recommendations are strictly adhered to. For Nigeria to meet this target of self sufficiency in beef production by 1986, efforts must be made to study and improve the reproductive performance of our beef animals.

Attempts must be sustained in improving nutrition or recovering more grazing area from the grip of tse-tse flies; improve management and control disease conditions limiting livestock production. In particular, research has to be centered not only on the pathology and pathogenesis of disease conditions but on the role of these disease causing agents on reproduction. One of such disease causing agents of economic importance in this country is Trypanosoma vivax.

Trypanosomiasis in Nigerian cattle is caused by 2 main trypanosome species viz: Trypanosoma congolense and Trypanosoma vivax. These trypanosomes together with other blood parasites produce acute, chronic and often fatal disease characterised by anemia and unthriftiness. The role of trypanosomes in limiting cattle production in Nigeria has been the object of many studies. Despite all the attention given to this pathogenic protozoon, very little attention or interest has been shown on the role of trypanosomes on bovine reproduction.

The objectives of this investigation therefore are to study the effects of:

1. experimental T. vivax infection on the fertility of heifers,
2. infection of T. vivax on first, second and third trimesters of pregnancy and
3. the immunoglobulin IgG and IgM response of pregnant heifers to T. vivax infection and to compare this response with that of non-pregnant heifers.

# REVIEW OF LITERATURE

## PART I

# REPRODUCTIVE PERFORMANCE OF NIGERIAN CATTLE

## Source of Information

Information on the reproductive performance of Nigerian cattle is rather scanty and based mainly on questioning and/or short-term observation (Fricke, 1964; FAO, 1966, 1975; Lamorde and Weinman, 1972; Lamorde and Franti, 1973; Akpokodje and Bolarinwa, 1974; Osori, 1976; Fullan, 1979) and from government farms records (Wheat and Broadhurst, 1968, 1972; Lamorde and Weinman, 1972; Nuru, 1974; Osori, 1976). Little information available for the Fulani animals that form more than 94% of the cattle population in Nigeria (Lloyd, 1976) is based mainly on surveys (Wheat and Broadhurst, 1968, 1972; Lamorde and Weinman, 1972, Nuru, 1974; Osori, 1976) and questionnaires (Lamorde and Franti, 1973 and Nuru and Dennis, 1976).

## Age at Puberty

Puberty is defined as that period of adolescence when a male or female is first able to release gametes (Hafez, 1975; Thibault and Levasseur, 1975) for example, first ovulation or estrus in the female. It is much more difficult to determine the exact time of puberty in the male because the first differentiation of spermatogonia precedes the release of the first spermatozoa from the seminiferous tubules by a month or more,

and sperm transport from tsetse to vas deferens needs at least 2 weeks (Thibault and Levasseur, 1975). However, at puberty, irrespective of sex, secondary sexual characteristics become conspicuous and the essential organs of reproduction begin to undergo great increases in size (Hafez, 1975).

The onset of puberty, is influenced by breed. (Wiltbank, 1978) climatic factors, social factors and nutrition (Oyedipe, 1981; Thibault and Levasseur, 1975) and involves changes at the pituitary level (Hammond, 1952). If nutrition is maintained at normal level, puberty is known to occur when body weight reaches 45% of the adult weight in cattle (Roberts, 1971).

Information on the age of puberty in cattle is rather scanty and sometimes with questionable accuracy. Asdell (1964) working with Zebu breeds in East Africa found that the age at puberty was 904 $\pm$ 43 days and in Nigeria, approximately 3 years in local Fulani heras and 2 years (22.3 months) in government farms (Lamorde and Weinman, 1972). In another survey by Nuru (1974) in the Northern Nigerian States, the age at first conception was between 3.3 - 3.7 years in nomadic cattle and 2.5 years in government farms. Data (Lamorde and Weinmon, 1972) show that in Shika Farm, in Nigeria, first estrus occurred between 7 and 8 months of age. These apparent earlier ages of reproductive maturity in Shika animals were attributed to genetic improvement

and management superiority of the farm over others. Recent studies by Oyedipe (1981), using Sahel animals have shown that nutrition plays a very significant role on the age at puberty. This worker found that heifers fed high protein diet achieve puberty at 570.4 days; heifers on medium protein diet at 640.8 days while heifers on low protein diet reach puberty at 704.2 days.

Reports from different parts of the world have shown that the Zebu - the predominent cattle breeds in Nigeria - reach puberty at 6-12 months later than the European breeds (Warnick et al., 1956; Temple et al., 1961; Reynolds et al., 1963 and Wiltbank et al., 1969). Similar observation has been made by Morrow (1968), Reynolds (1972) and Devaccaro et al., (1977). These workers used the Brahman - a Zebu type cattle for their comparison with the Bos taurus breeds. This late age of attaining puberty is seen as serious economic problem in our cattle industry and it has been observed by Wiltbank (1978) that puberty is a genetic problem in Zebu and Zebu type heifers.

Age at First Calving

Age at first calving is a fertility index and is closely related to the age of puberty. However, there are several factors affecting the heifer and causing the age at first calving to be quite unrelated to the age at puberty. Such factors include early embryonic death,

anestrus and conception failures of various etiology. Thus age at first calving is here reviewed seperately from the age at puberty.

Information on the age at first calving in our indigenous Zebu breeds of cattle is rather scanty. Where the information does exist, it is mainly from government herds especially the livestock improvement and breeding centres (LIBC) and little or none for the numerous herds kept by the nomadic Fulani.

Croix (1944) noted that heifers in Northern Nigeria calved before they were 4 years old. Fricke (1964) reported that the age at first calving for the White Fulani cattle in Northern Nigeria was between 3½ to 5 years. Wheat and Broadhurst (1968) and Wheat (1972) found that the age at first calving for the White Fulani cattle, Rahaji, Azaouak and Sokoto Gudali breeds kept in LIBC's ranged between 33-47 months. In a similar report, Knudsen and Schael (1970), working in a government farm in Vom put the age at first calving of the Zebu at 45 months. Using data from various sources, Zemjanis (1974) put the average age at first calving of heifers from nomadic herds at 60 months and those from government herds at 40 months. In their own study, Nuru and Dennis (1976) gave 4½ years as the age at first calving. Working with White Fulani cattle kept in the southern environment, Akpokodje and Bolarinwa (1974) found that the average age at first calving was 46.55 months. Similar studies have been conducted by

Pullan (1979) and the age at first calving was found to be 5 years. In one herd, however, Pullan (1979) found that the average age at first calving was 40 months. This was shown to be similar to figures from Vom government farm where the age was 39 months. Thus it can be said that the age at first calving among our indigenous cattle breeds ranges from 39 months in the well-managed government farms to 60 months in the nomadic cattle herds. In all, improved management, intensive system and genetic up-grading of the animals were shown to affect the age at first calving.

Similar studies with Zebu and Zebu type cattle from other parts of Africa show similar results. In East Africa, Mahadevan (1965), calculated the average age at first calving for the Ngauda, Boran and Jiddu and found they were 42, 43 and 40 months respectively. A range of $3\frac{1}{2}$ to $4\frac{1}{2}$ years has been reported from Botswana (Reed et al., 1974) while in the Sudan, the age at first calving ranges from 30 to 96 months (Osman, 1972). In the Bos taurus breeds of cattle, the mean age at first calving has been put at 30 months (Roberts, 1971).

Calving Interval

Calving interval is the period between two successive calving and is a useful fertility index for assessing the reproductive performance of a herd.

Extended calving intervals may be caused by all major forms of infertility including anestrus, conception failure and prenatal death (Osori, 1976).

There is very little information on the calving interval of our indigenous cattle in the nomadic herds. Most of the information we have for these groups of animals is based on surveys and questionaires. Fricke (1964) reported an overall figure of 16 months as the calving interval of the While Fulani cattle in northern Nigeria. Information from Nuru's survey (1974) shows that calves nursed for up to 10-12 months and that the first estrus occurred at about 15 months post partum. Thus, even if conception occurred with this estrus, the calving interval would then be 24 months. Similar figures were quoted by Lamorde and Franti (1973). Akpokodje and Bolarinwa (1974) working with White Fulani cows kept under semi-intensive system in [illegible] southern environment, reported a calving interval of 454.9 days.

Wheat and Broadhurst (1968, 1972) calculated the calving interval for the indigenous breeds of cattle in government farm in Nigeria. For the White Fulani, the calving interval was between 388 and 463 days while that of Sokoto Gudali was between 439 and 496 days. Wheat (1972) reported the calving interval of the Azaouak and Rahaji to be between 423 and 537 days. Using records from Shika Farm, Zemjanis (1974) calculated the calving

interval of Nigerian indigenous breeds to be 447 days. Pullan (1979) reported that the calving interval for cows in the government farms were lower than those in the nomadic herds. The interval ranged from as low as 12 months for a government farm at Vom to as high as 27 months in the nomadic herds.

A look at the calving interval for Zebu breeds outside Nigeria shows similar figures. In East Africa, Mahadevan (1965) reported a calving interval of 420 days for the N'dama breeds, 362 days for small East African Zebu and 382 for the Boran. In central Africa, Rakha et al., (1970) reported calving interval of 379 and 425 days for the Angoni and Africander breeds respectively.

Annual Calf Crop

Calf crop is defined as the number of calves produced during the year by cows and heifer of reproductive age expressed as a percentage. Annual calf crop has been shown to be an important factor in the efficiency of production (Lasley, 1972). It is a measure of the reproductive performance of a herd.

Here in Nigeria, there is only scanty information on the calf crop in the Zebu cattle. Most of this information is based on surveys and limited data from records in government herds (Osori, 1976). Wheat and Broadhurst (1968 and 1972) have shown that the annual

calf crop of Nigerian indigenous Zebu cattle is about 60 percent. The survey by Lamorde and Weinman (1972), showed that the annual calf crop among the indigenous herds varied between 27 and 55 percent. In a similar study, Nuru (1974) found that the annual calf crop among indigenous herds was between 34 and 47 percent. The same worker also found that from the 10 LIDC's he surveyed, the annual calf crop was 67.2 percent. This observation was similar to that of Johnson (1974) who reported that the annual calf crop of White Fulani cows in Shika was 62.1 percent. Working with very limited number of cows in Vom, Schael (cited by Osori, 1976) reported an annual calf crop of 100 percent.

In a similar study, using the zebu and zebu type animals outside Nigeria, Rakha et al., (1970) found that the annual calf crop for the Angori, Africander, Mashona were 83%, 65% and 83% respectively. These were said to be above the national average and the higher rates were attributed to the improved nutrition. In Nyasaland, Wilson (1946) reported an annual calf crop of 27.5 - 31.2 percent. In Botswana, the annual calf crop is between 39.8 and 51.6 percent (Reed et al., 1974).

Other Indices for Reproductive Performance

In more advanced countries, reports abound on post partum in-activity period, generation interval, number of services per conception and non return rates. Here, such reports are almost non existent. In the White Fulani and White Fulani Friesian cross, Oyedipe et al., (1981) reported that the average period of post partum inactivity was 16.5 and 21.4 days respectively. A generation interval of 72 to 75 months has been reported for the indigenous cattle (Wheat and Broadhurst, 1968).

The available survey information on the reproductive performance of indigenous zebu cattle has been summerised by Zemjanis (1974) as follows:-

| | Nomadic cattle | LIBC's |
|---|---|---|
| Age at first calving (months) | 60 | 40 |
| Calving interval (months) | 17-24 | 16-18 |
| Annual calf crop (%) | 40 | 60 |
| Total no. of calves produced | 2½ | 4 |

The Reproductive Pattern of the Nigerian Zebu

The reproductive pattern of the Zebu cattle of Nigeria has been the focus of many studies (Wheat and Broadhurst, 1972; Lamorde and Weinman, 1972; Lamorde and Franti, 1973; Osori, 1976 and Kumi-Diaka et al., 1981). Most of these studies have been based mainly on limited observations. The validity is similarly limited.

Evidence of seasonality in the reproductive pattern of Zebu cattle of Nigeria comes mainly from the reports of Lamorde and Weinman (1972) and Lamorde and Franti (1973). According to these reports, most calving here occur mainly during the early wet season. Earlier observation had been made by Steinbeck and Balogun (1971). Using records from a government herd in Ibadan, these workers found that the Keteku cattle peak period of sexual activity varies between February and June. However, other workers (Wheat, 1972; Wheat and Broadhurst, 1972; Zemjanis, 1974) have shown that this peak varies between the different Zebu breeds and vegetation location in Nigeria.

A more detailed clinical and histological 15-month study by Osori (1976) on the reproductive pattern of the Zebu cattle in Nigeria showed that there were seasonal variations in the number and size of Graafian

follicles, ovarian size, weight and cyclic activity. According to this study, ovarian function peaked between July and August. It was then concluded that indigenous cows in Northern Nigeria are seasonal breeders with peak activity during the rainy and pre-dry seasons. This seasonality is closely related to the availability of feed (Osori, 1982, personal communication). Similar observation has been reported by Ojo et al. (1977).

Reports from other parts of Africa indicate that the Zebu cattle have peak periods of sexual activity (Osman, 1972; Wilson, 1946). Osman (1972) working in the Sudan observed that cows which calved during the rainy season (July - October) required more services per conception than those calving during the dry season (March - June). In central Africa, Wilson (1946) reported that calving was seasonal and mainly occuring between June and September.

Rakha and Igboeli (1971) studied 40 cows, 20 on normal and 20 on low planes of nutrition. They observed no seasonal effects in either the estrous cycle or calving in the group with normal nutrition. In the second group, cycling ceased even before the end of the experimental period. Records here in Shika and the University Farm of Ahmadu Bello University also show no seasonality in the reproductive performance of the Zebu

(Unpublished data from Theriogenology Section of the Department of Veterinary Medicine and Surgery, Ahmadu Bello University).

## BOVINE ABORTION

Abortion defined as the expulsion, before full terms, of a conceptus incapable of independent life differs from premature delivery which is the expulsion before full term of a fetus capable of independent life (Hubbert, 1971).

From the above definition fetal death is not an essential prelude to abortion though most reported cases involve dead fetuses. Many fertilized ova, embryos or early fetuses may be aborted without being seen especially when the animals are on the range and not closely observed (Roberts, 1971). Abortions are usually caused by factors, for example stress, or agents (both infectious and non-infectious) affecting the fetus or its membranes.

There seems to be scanty information on the variations in hormonal levels during abortion in animals. Osburn et al., (1969) and Adelakoun et al., (1978) reported a decline in the serum progesterone levels on the day of abortion similar to that seen in normal parturition.

The later authors found the estrone levels lower in aborting cows than in calving cows. Estradiol-17-B levels were also low in aborting cows, and the gradual

increase throughout pregnancy characteristic of calving cows was conspicuously absent. The decline in sex steroid levels may be associated with placentitis as well as luteolysis in the case of progesterone.

Severe and prolonged maternal stress may be associated with abortion or premature delivery (Osburn and Kennedy, 1981). According to these authors, maternal corticosteroids when present at levels of 100 to 120ng/ml for 48 hours may eventually raise fetal blood levels to 20 to 40ng/ml and this is sufficient to cause abortion. The corticosteroids cross the fetoplancental unit sufficiently to decrease progesterone and increase estrogen and prostaglandin production (Hafez, 1975).

In humans, hormonal changes have been used in the surveillance of highrisk pregnancies (Goebel, 1978). If the production rate of human chorionic gonadotrophin (HCG) and human placental lactogen (HPL) decrease, the likelihood of an unavoidable abortion is extremely high (Gerhard and Runnebaum, 1977). The same is true when the levels of ovarian hormones (estrone, estradiol-17-B and progesterone) induced by HCG and are necessary for maintenance of pregnancy drop (Tulchinsky and Hobel, 1973).

The reported incidence of bovine abortion based on limited observations ranges from 7 to 12 percent of those cows and heifers found to be pregnant between 30 to 50 days after breeding (Holt, 1952; Withers, 1957; Mitchell, 1960; Miller, 1980).

It should be pointed out that the exact figures regarding abortion are not available not even in the sophisticated societies of the world. A 4-year-study by the British Research Council (cited by Frank, 1963) reported that 6.5 percent of all calves were born dead and that 56 percent of losses of calves up to 6 months of age were prenatal. In the same report, Frank, (1963) cited the studies carried out in New Zealand and America. In 1956, the New Zealand Dairy Board cited 2.8 percent incidence of abortion and added that only half of the abortions were actually reported. In the survey conducted by the Research Committee of America Cattlemen's Association, it was shown that only 80 percent of 20,000 cows studied producted live calves and 62 percent weaned calves (Frank, 1963).

A more recent report from the American National Academy of Science cited by Hubbert et al., (1973) indicate that the annual losses in the United States calf crop were between 14 and 36 percent. These estimates included prenatal losses of embryos and fetuses

(abortion, stillbirths, congenital malformations) as well as postnatal loss of calves.

Here in Nigeria, there are few reports on the incidence of bovine abortion. The survey conducted by Lamorde and Weinman (1972) indicated a 12.4 percent abortion rate amongst Fulani herds and 3.9 percent in Goverrment farms. In the study by Ogwu et al., (1982), 112 fetal deaths were diagnosed clinically in the different farms with a total cattle population of 3,300. Experience in our laboratory shows that the incidence of abortion in some Government farms is as high as 20%. Our experience also suggest that the incidence of abortion is higher in Government herds than in the Fulani herds.

The disparity in the abortion rates is purely due to management systems. This observation has in fact been confirmed by Nuru and Dennis (1976) who reported that the lowest incidence of abortion was in local herds while the government herds have the highest incidence of abortion.

This is mainly due to the semi-intensive system of husbandry practised in the Government farms which tend to concentrate the flock in a small area as opposed to the extensive system practised by the Fualni's thus reducing chance of infection.

## CAUSES OF BOVINE ABORTION

Several workers (Osebold et al., 1960; Dennis, 1969; Hubbert et al., 1973) have shown that abortion is still one of the most puzzling aspects of reproductive failure. Although notable progress has been made in determining the cause of bovine abortion, reports show that over 70 percent of the abortion fall into no recognised diagnostic category (Howarth et al., 1956; Dennis, 1969). Working with 3,812 bovine abortions spread over 11 years period, Hubbert et al., (1973) could not identify the etiological agent in 76.7 percent of the cases. Among the diagnosed cases of abortion, a variety of agents have been incriminated. These include: physical, genetic or chromosomal, nutritional, chemical, drug or toxic and infectious causes (Roberts, 1971).

### P H Y S I C A L

Bovine abortions resulting from physical causes are rare (Robert, 1971). Before abortion can result from physical causes, there has to be severe trauma to the dam or the conceptus. Ball and Caroll (1963) found that manual rupture of the amniotic vesicle in early pregnancy (30 to 60 days) will terminate pregnancy. The full impact of this observation can only be appreciated when one reflects on the fact that pregnancy diagnosis

in cattle is done by rectal palpation not only of the pregnant uterus but also of the amniotic vesicle especially in very early pregnancies.

Thus there have been questions about the possibility of abortion as a consequence of uterine palpation (Cowie, 1948; Rowson and Tott, 1963; Ball and Carroll, 1963, Zemjanis, 1970). Several other investigators have shown that abortion can be induced when excessive pressure is applied to the amniotic vesicle or to the conceptus (Ball, 1968; Dawson, 1974; Parmigiani et al., 1978).

Douching the uterus by invading the cervix with a pipette or catheter and introducing intra-uterine medication such as Lugol's solution and for the purposes of insemination have been recorded as physical causes of abortion (Roberts, 1971). Other physical causes of abortion include severe torsion of the uterus, displacement of the umbilical cord over the head and neck of the fetus or torsion of the umbilical cord resulting in fetal death.

## GENETIC OR CHROMOSOMAL

In humans, a number of studies have implicated chromosome anomalies as an important cause of reproductive wastage (Carr, 1967). More than 20 percent of spontaneous abortions have been associated with chromosomal abnormalities (Carr, 1967). Studies on embryos of farm animals suggest that chromosome anomalies may contribute significantly to fetal mortality although the exact nature and extent of involvement is not known (McFeely, 1976).

Since the pioneering work of Gustavsson (1969) on autosomal translocation in Swedish cattle, a lot of other chromosomal anomalies have been discovered in other European breeds of cattle. These include chromosome breaks and achromatic gaps in addition to 1/29 translocation in Canadian Guernsey bulls associated with reduced fertility (Bongso and Basrur, 1976); Robertsonian translocation in British Friesian cattle (Pollock, 1972, Pollock and Bowman, 1974), metacentric fusion of chromosomes 3 and 4 in Limousine (Popescu et al., 1977), centric fusion in Swiss Simmental breed (Popescu et al., 1975), pericentric inversion in Norman cattle (Popescu, 1972; Popescu, 1976), reduction in chromosome number due to centric fusion in Norwegian Red cattle (Amrud, 1969) autosomal translocation in Charolais (Harvey, 1971) and Holstein-Friesian (Herschler and Fechheimer, 1967).

The role of chromosome anomalies in embryonic and fetal mortality has not been fully determined. Several investigations (Refsdal, 1976; Pollock, 1972; McFeely, 1976; Malnan, 1972; Gustavsson, 1971, Bongso and Basrur, 1976) have shown that these anomalies result in infertility. Serious losses of potential productivity through structural and numerical changes arising de novo in embryoes have also been attributed to chromosomal aberrations (Fechheimer, 1972). These changes result in early embryonic death or abortion. Polyploidy is rare in postnatal individuals. However, working with blastocysts, McFeely (1968) found several polyploid bovine conceptuses. These zygotes are therefore abnormal and embryonic deaths, fetal resorptions and abortions are natures way of getting rid of abnormal zygotes.

Autosomal recessive gene action has been reported as a cause of abortion in a Holstein herd (Steven and King, 1969). These investigators reported the abortion and fetal mummiffication occurring at 120 to 180 days of gestation. The fetal deaths occurred in the daughters of 2 popular sires. Inbreeding is reported to cause embryonic death, abortions and stillbirths by the concentration of lethal genes in a conceptus (Mylrea, 1963, Roberts, 1971).

## NUTRITIONAL

The response of animals to their food supply is mainly reflected in their reproductive process. If food supply is low, reproduction is either greatly reduced or ceases (Roberts, 1971). Nutritional factors are more commonly associated with infertility than with abortion (Miller, 1980). However, according to Noustgaard (1969), if during pregnancy malnutrition happens to coincide with the first third of gestation, that is, during the sensitive periods of organogenesis, development of malformations and even fetal death may be a sequela. In the fetal stage (second and third trimesters) such deficiency may result in malformations mainly in such organ systems as have several sensitive functions. In extreme deficiency, death may ensure. Roberts (1971) reports that acute, severe starvation may result in abortion and that this acts as a protective mechanism to preserve the maternal animal's own body reserves.

Selenium-vitamin E deficiency has not been definitely established as a cause of cattle abortion (Gibbons, 1964). However, Hastings (cited by Faulkner, 1968) reports a condition manifested by the birth of dead, weak or premature calves and a high incidence of retained placenta that appeared to respond to vitamin E and selenium injections.

Iodine deficiency as a cause of abortion has been reported (Allcroft et al., 1954; Moustgaard, 1969). These investigators have also attributed the birth of weak, hairless, stillborn or goitrous calves to iodine deficiency.

Vitamin A deficiency adversely affects reproduction in all species (Robert, 1971) including bovine abortion usually late in gestation (Gibbons, 1964; Thomas, 1968) and birth defects involving the eyes. Vitamin A deficiency causes keratinization of the vaginal epithelium and placental degeneration leading to abortion (Roberts, 1971). Blood serum levels of vitamin A in cattle at the time of abortion or shortly after are less than 25% mcg/100ml and frequently are 12 - 15 mcg/100ml provided feed intake has not been increased (Gibbons, 1964).

There is no evidence that other vitamin, mineral, protein or other nutritional deficiencies will cause abortion in cattle (Roberts, 1971).

## CHEMICAL, DRUG OR TOXIC

Toxic substances especially those in plants are frequently suspected abortifecients but are usually difficult to document (Miller, 1980). Several chemicals for example nitrate poisoning, drugs and phytotoxins are known to result in bovine abortion (Simon et al., 1958, 1959; Winter, 1968; Roberts, 1971). Simon et al. (1958) described the pathological changes associated with nitrate abortion. In the report, the authors stated that up to 80 percent of the abortions occurred in the herd from 3 to 9 months of gestation. The abortions occurred when the cattle grazed on improved pasture high in nitrate content. Low levels of the chemical are reported to be unable to produce abortion (Winter and Hokanson, 1964; Davidson et al., 1964; Crawford, 1960; Winter, 1968). Winter (1968) stated that nitrate intoxication does not cause abortion per se in healthy ruminants receiving balanced ration. It is the authors opinion that under yet undefined circumstances, nitrate or its reduction products may act in conjuction with other factors to produce abortion.

Other chemical causes of bovine abortion include: chlorinated naphthalenes which among other things produce hyperkeratosis (X disease) an acute vitamin A deficiency, metaplasia of the epithelium of many internal

organs, metritis and abortion (Hansel and McEntee, 1955) and arsenicals (Udall, 1954). The role of lead, sodium iodine, sodium chlorate in bovine abortion is questionable (Roberts, 1971).

Some phytotoxins when ingested in sufficiently large quantities may result in abortion. Of interest here are the pine needle and locoweed abortions.

## INFECTIOUS ABORTION

Although the cause(s) of bovine abortion remain undetermined in approximately 70 percent of reported cases (Howarth et al., 1956; Osebold et al., 1960; Dennis, 1969; Hubbert et al., 1973; Kirkbridge et al., 1977), infection remains the single most important cause (Hubbert, 1973; Miller, 1980; Osburn and Kennedy, 1981). One of the reasons for this is the fact that compared to humans, domestic animals are often very young when they first become pregnant. The hazard of systemic infection of the dam during pregnancy is increased by indiscriminate movement of the animals which in turn makes it more likely that an individual animal will be exposed for the first time to an infectious agent (Osburn and Kennedy, 1981).

Some of the infectious agents may produce disease in both the fetus and the dam while in others the consequence of dam infection is quite different from that of the fetus. This is due mainly to the fact that while the dam is more matured and has sophisticated systems for handling infectious agents, the bovine fetus is largely agammaglobinemic, specific microbial growth factors (erythritol for Brucellanceae) are present and the rapidly dividing cells that are needed for replication of some viruses are present in numerous

fetal organs (Osburn and Kennedy, 1981). These factors favour environment for the microbes to proliferate with minimal hindrance.

It has been established that all infectious abortion-producing agents result in placentitis (Dennis, 1969). Thus although gross and microscopic lesions found in aborted fetuses are not always diagnostic, placental lesions may be pathognomonic, suggestive or non-specific (Dennis, 1969). Many people (Dennis, 1969; Njoku et al., 1973; Pier et al., 1972).seem to agree that placentitis is a common finding in all cases of infectious abortion. However in abortion associated with piroplasms (Christie, 1963; Klinger and Ben-Yossef, 1972; Fowler and Swift, 1975; Swift and Paumer, 1978; Correa et al., 1978; Swift, 1979) and trypanosomes (Nuru, 1974), no placentitis were reported. Thus placentitis may not be a necessary prelude to all infectious abortions. The mechanism of abortion in piroplasms has been attributed to genital fetal infection (Correa et al., 1978), anoxia and accompanying acidosis from maternal anemia (Swift and Paumer, 1978). The mechanism of abortion due to trypanosomes is harder to explain. It is speculated that transplacental transmissions do occur and abortion is due to fetal infection even though the parasite may not be easily demonstrated in the fetus (Ogwu and Nuru, 1981).

Bacterial and mycotic agents produce abortion in the bovine mainly by the production of toxic products - endotoxins in gram-negative bacteria (Reider and Thomas, 1960; Dennis, 1961). Most gram-positive bacteria result in fetal infection and death and subsequent abortion producing uterine and placental lesions (Njoku et al., 1973; Njoku and Dennis, 1973.

Viral infections often result in fetal malformations or dysplasia (Sever and London, 1969; Ward, 1969; Bistner et al., 1970; Elizan and Fabiji, 1970; Kahrs et al., 1970a; Kahrs et al., 1970b; Kahrs et al., 1970c; Kahrs et al., 1971; Catalano and Sever, 1971; Scott et al., 1973; Osburn and Kennedy, 1981) resulting often in the death and abortion of the infected fetuses. Persistent infection occurs with certain viruses such as blue tongue resulting in the calves becoming a potential reservoir of infection throughout life (Osburn and Kennedy, 1981). In others like I.B.R., there is extensive cell destruction in all stages of gestation but apparently abortion occurs most frequently in the last trimester (Kennedy and Richards, 1964; Kelling et al., 1973; Zemjanis and Hurtgen, 1975).

## BOVINE ABORTIONS IN NIGERIA

There are very few reports on bovine abortion in Nigeria. The available reports suggest that it is sporadic (MacGregor, 1962; Lamorde and Weinman, 1972; Lamorde and Franti, 1973; Nuru, 1974) and that the livestock owners tend to live with it without informing the Veterinary authorities (Nuru, 1974). The early reports from Veterinary Division of the Ministry of Health and Forestry Resources of Northern Nigeria 1959 - 1963 indicate that abortions were confined to the then Benue Plateau and North-Eastern States (MacGregor, 1959, 1960, 1962, 1963). Early sporadic abortions in cattle were also reported in Kontagora, Bida and Minna Agricultural Centres (Anon, 1960). In the late 1950's, both Adamawa and Zaria provinces reported that bovine abortion in the areas were of considerable economic and public health significance (Anon, 1956). Abortions and breeding difficulties were reported at the Birnin Kudu and the Raav LIBC (MacGregor, 1962).

Reported incidence of abortion in herds varied from state to state. In local herds, it ranged from 0.9 percent in North-western state of 43.1 percent in North-eastern state (Lamorde and Franti, 1973 and Nuru, 1974). Lamorde and Weinman (1972) reported a 12.4 percent abortion rate in local Fulani herds and

3.9 percent in Government farms. Nuru (1974) found that abortion was more widespread in the savanna grazing areas of the country than was previously thought.

Although the bulk of bovine abortions in Nigeria remain undiagnosed most official reports tend to incriminate brucellosis and trypanosomiasis as the major etiologic agents of bovine abortion (MacGregor, 1962). Nomadic Fulani cattle owners consider trypanosomiasis as an important bovine abortifacient coming second only to nutritional deficiencies (MacGregor, 1962; Lamorde and Franti, 1973, Nuru, 1974).

Bovine Brucellosis in Nigeria

Brucellosis has been reported sporadically from various parts of the country since 1927 (Banerjee and Bhatty, 1970). Hygroma or "Bokkale" in cattle is well known to the Fulani cattle owners and has been associated with bovine abortion (Anon, 1957). This observation has in fact been confirmed by the high brucella titre in the "bokkale" sera (Nuru, 1974) and by the isolation of Brucella abortus organism (Fensterbank, 1978). Bovine brucellosis (Bang's Disease) is caused by a small gram-negative rod, Brucella abortus, that grow intracellularly (Roberts, 1971). It was first described in Denmark by Bang in 1897 and the organism was first isolated in Uganda by Bruce in 1910 (cited by Kagumba

and Naudokha, 1978). Brucellosis is the most important worldwide cause of abortion in cattle except in the U.S. where the eradication program has been most successful (Roberts, 1971). Brucella abortus causes abortion in cattle in the last trimester and a subsequent period of infertility is usually experienced. The transmission, pathogenesis, clinical signs and diagnosis of infection is very well established. (Lambert et al., 1960; Manthei, 1968; Blood and Henderson, 1968; Roberts, 1971; Alton et al., 1975).

Our knowledge of bovine brucellosis in Nigeria is based mainly on the sporadic serological surveys (Adams and McKay, 1966; Banerjee and Bhatty, 1970; Nuru, 1974; Esuruoso, 1974a; 1974b). There are only few reports on the actual isolation of the brucella organism (Eze, 1978; Bale, 1980).

Working with government herds in Northern Nigeria, Banerjee and Bhatty (1970) found that the incidence of bovine brucellosis in 1934 was 15%; 9% in 1946; 30% in 1947 and 11.8% in 1948. Serological results of cattle bled at various parts of Sokoto province (State) in 1970-71 showed that the prevalence rate for Bulassa was 12%; 15% for Dogon Daji ranch; 20% for Talata Mafara and 0% for Kotor Kushi LIBC (Bid, 1972, cited by Nuru, 1974). In a similar serological study

of cows in the then six Northern states, Nuru (1974) found the highest rate of brucella infection in Kwara state 26.4% followed by North-eastern state 3.3%; North Central 0.7% and 0.0% in Kano state. The reported infection rate in the Fulani cattle in the six Northern state was 7.1% and 3½ 3.6% in a government ranch (Nuru, 1974).

In the Western state, N'dama and Keteku cattle have been reported to be the most heavily infected (Esuruoso, 1974b). It was pointed out by this author that these herds were the breeding units from which new herds derived their stock. An infection rate of 23.3% was reported in the Keteku herd at the Upper Ogun distribution centre. In Bendel state (Mid-West) 28.0% infection rate was reported for the Igara ranch; 12.5% for the Ubiaja ranch and 31.03% for the Oria ranch (Esuruoso, 1975). In the Eastern states, Adams and McKay (1966) reported 1.2% infection rate in a government herd of zebu cattle in 1962. A more recent report from Nsukka (Anambra State) indicated a 10% infection rate in a herd of N'dama cattle located at the University of Nigeria, Nsukka teaching and research farm (Esuruoso, 1975).

## OTHER INFECTIOUS ABORTIFACIENT IN NIGERA

Alhough brucellosis and trypanosomiasis are generally regarded as the major infectious abortifacient diseases in Nigeria (Nuru, 1974), there are others such as leptospirosis that are known to be present here whose association with bovine abortion have not been well investigated.

Leptospirosis: In most parts of the world, the Hebdomadis serogroup and in particular the Hardjo serotype has been recognised as a cause of abortion and infertility in cattle (Amatredjo and Campbell, 1975). Ellis and Michna (1976) have demonstrated a relationship between the presence of infection in cattle by the Hebdomadis serogroup of leptospira and a history of undiagnosed abortion in Scotland. These workers also isolated a leptospira of this serogroup from an 8 month fetus. This association of leptospirosis with bovine abortion has also been established by Little et al., (1980) and Higgins et al., (1980).

In North America, Leptospira pomona appeared to be the principal serotype involved in bovine abortion (Boulanger et al., 1958; Hanson, 1960; Stoenner, 1968), but the L. grippotyphosa and L. hardjo have also been incriminated (Hanson and Brodie, 1967; Diesch et al., 1971; Roberts, 1971).

Abortion occurs in the last trimester and within 6 weeks after the acute phase (Roberts, 1971). The abortion rate varies from 5 to 40% depending upon the number of susceptible cattle in the last rimester of gestation, virulence of the leptospira, and the opportunity for spread of infection (Stoenner, 1968). Not all susceptible infected cows in advanced pregnancy abort; occasionally, an infected cow may give birth to a weak, non-viable calf that dies within a few days (Roberts, 1971, Ellis and Michna, 1976). Retained placenta, metritis and infertility are common sequel of leptospiral infection in cattle (Stoenner, 1968). Other clinical signs include sudden drop in milk yield, fever hemoglobinuria, anaemia, jaundice and death (Roberts, 1971).

A fetal leptospiraemia occurs following invasion of the placentomes and fetal death is considered to be due to anoxia from leptospiral haemolysin (Fennestad and Borg-Petersen, 1958). The fetus is usually dead for about 24 hours or more before expulsion; consequently the lesions are non-specific and are due to in-utero autolysis viz: generalized hemoglobin inhibition in all tissues, blood tinged subcutaneous edema and effusions in the serous cavities (Dennis, 1969). The uniform edematous and autolytic involvement of the placenta is suggestive of leptospirosis (Dennis, 1969). The inter cotyledonary areas are edematous and semitransparent

with brown gelatinous fluid between the amnion and allantois (Te Punga and Bishop, 1953). All cotyledons are light tan to yellowish, atonic and avascular (Dennis, 1969).

Here in Nigeria, Eze (1981) has shown that there is a high prevalence of leptospiral antibodies in our cattle population. In his study on eight hundred cattle examined serologically, he found 63% positive at serum dilutions of 1:100 and above. Turner et al., (1975) had earlier obtained a 63.9% positive by microagglutination test to L. grippotyphosa, L. hardjo and L. pomona.

Although these workers have shown evidence of high prevalence of leptospiral titre, there are no reports of the association of leptospirosis with bovine abortion in Nigeria. However, the Theriogenology Section of this Department did investigate a case in Kaduna State and found that leptospira organism was responsible for the abortion. Recently also the Section was involved in an unconfirmed case of leptospiral abortion in a government herd in Kano State.

## VIRAL ABORTIONS IN NIGERIA

Although viral agents such as I.B.R., P.V.D., and $PI_3$ have been associated with bovine abortion (Chow et al., 1964; Sever and London, 1964; Kendrick and Straub, 1967; Chow, 1968; Kahrs et al., 1970a, 1970b, 1970c; Roberts, 1971; Kendrick, 1971; Kelling et al., 1972; Hubbert et al., 1973; Swift, 1973; Zemjanis and Hurtgen, 1975), very little is known about them here in Nigeria (Zwart, 1966; Taylor et al., 1977) and their association with bovine abortion in Nigeria remains unknown. Using serology, Zwart (1966) was able to demonstrate the IBR virus was widespread in the Nigerian cattle population even though the disease in its clinical manifestations has never been identified. Taylor et al., (1977) has shown that there is a widely scattered BVD virus antibody in sheep and goats in Nigeria. In a similar study by Taylor and Pullan (cited by Taylor et al., 1977), 44 percent prevalence rate was recorded in cattle. There was no association of these viruses with bovine abortion.

## BOVINE TRYPANOSOMIASIS IN NIGERIA

Bovine trypanosomiasis is another abortion-causing disease in Nigeria. For years the settled Fulani cattlemen have observed that the Federal Government's sponsored tse-tse fly eradication campaign and the use of adequate chemotherapy had greatly reduced the abortion rate in their herds. The association of trypanosomiasis with bovine abortion has been based on rather circumstantial evidence (Kirkby, 1963; Maclennan, 1963; 1970; Leefland, 1975; Nuru, 1974).

In reviewing the occurrence and epizootiology of bovine trypanosomiasis in Shika farm, Kirkby (1963) observed that the high abortion rate in 1948 was associated with T. congolense infection as no other abortifacients were found. In that year a 16 percent infection rate was recorded which subsequently increased to 31 percent infection rate in 1958 with 59 out of 131 cattle (31%) aborting. In 1972, 8 of 10 aborting cows in the same Shika farm were infected with trypanosomes. In 1973, 2 out of 3 cows that aborted in Shika farm had trypanosome infection (Leefleng, 1975). So important is the trypanosomiasis problem in this country that it would be necessary to look at trypanosomes and cattle production in Nigeria.

## TRYPANOSOMES AND CATTLE PRODUCTION IN NIGERIA

Trypanosomiasis continues to be one of the most important obstacles to cattle industry in Nigeria, despite the efforts of the Federal Pest Control Department of the Ministry of Agriculture. The wide-spread distribution of tse-tse flies - the vectors of African trypanosomiasis has imposed a nomadic or transhumance system of livestock husbandry on the Fulani cattle rearers (Loeflang, 1978).

In a study conducted by the World Bank and Nigerian consultants for the Federal Ministry of Agriculture and the National Committee on Green Revolution (1981), it was estimated that 600,000 to 700,00 sq. km or 75% of the entire country, is infested with the tse-tse fly. This, according to the study, includes 25% of land infested seasonally and 50% infested throughout the year. It is clear therefore that tse-tse flies and trypanosomes are important in the Nigerian livestock industry.

The importance of the disease was recognised by the colonial administration in Nigeria which in 1947, set up the West African Institute for Trypanosomiasis Research (WAITR) in Kaduna at a relatively high investment cost of £350,000.00, David West (1981) noted that

an investment of this colossal sum was in recognition of the importance of the disease. Today, the Nigerian Institute for Trypanosomiasis Research (NITR) which replaced the WAITR together with the Federal Pest Control Department, the Faculties of Veterinary Medicine throughout the country and other institutes and organisations have contributed immensely to the control of the menace of trypanosomiasis in our livestock. Despite their combined efforts however, trypanosomiasis still remains a major obstacle to livestock production in Nigeria (Ikede, 1981).

The apparent failure to control trypanosomiasis despite technological developments in medicine has been attributed to the highly variable nature of bovine trypanosomiasis (Mansfield, 1978; Tizard et al., 1978a). Secondly, it is only in recent years that the disease and organisms have been studied with relatively sophisticated techniques (Tizard et al., 1978b).

The group of monomorphic salivarian trypanosomes is by far the most important to the livestock industry (Leefland, 1978). This include two species: Trypanosoma congolense and T. vivax. T. vivax is probably the most common trypanosome in Nigerian cattle (Glover, 1961). T. congolense is the most important cause of bovine trypanosomiasis in East Africa (Tizard et al., 1978).

T. brucei, the third important trypanosome of cattle, is the least pathogenic for the domestic animals (Hornby, 1949; Kenning, 1956; Stephen, 1970; Lizard et al., 1978) though it has been reported that up to 30% cattle in some regions of Nigeria may be infected with T. brucei (Godfrey and Killick-Kendrick, 1961; Godfrey et al., 1964; Gray, 1970). The non-pathogenic T. theileri is widespread in Nigeria and 60-100% of our cattle may be infected (NITR, 1964).

Clinical signs of bovine trypanosomiasis resemble those seen in human trypanosomiasis and include gradual but progressive onset of irregular fever, anemia, splenic, hepatic and lymph node enlargement and cachexia (Apted, 1970; Ormerod, 1970). Affected individuals eventually show neurological disturbances including hyperesthesia, lassitude and weakness. Loss of muscle control, somnolence, coma and death may also occur except that neurological signs are rare in animals (Stephen, 1970). The signs in animals include intermittent fever, extreme anemia, lymphatic enlargement hepatosplenomegaly. Progressive cachexia involving both body fat and muscle has been regarded by most researchers as a very significant feature at least from the standpoint of the economics of livestock production. The course of the disease is highly variable and ranges from the apparent asymptomatic carrier state to a very

severe acute disease with death within a few weeks (Apted, 1970; Ormerod, 1970).

The distribution of trypanosomes within affected animals depends on the species of trypanosome involved. Trypanosomes of the T. brucei group (T. brucei brucei, T. brucei gambiense and T. brucei rhodesiense) is capable of penetrating the capillary tissues (Losos and Ikede, 1970; Godwin and Guy, 1973) and of multiplying outside the blood vascular system. It is therefore not surprising that this group of trypanosomes are widely distributed outside the vascular system and often encountered in cerebrospinal fluid, aqueous humor, lymph and in connective tissue fluid (Hornby, 1949; Fernhead et al., 1974). Trypanosoma vivax and T. congolense, on the contrary, do not usually leave the blood stream (Losos and Ikede, 1973; Losos et al., 1973; Tizard et al., 1978). They tend to lodge preferentially within the microcirculation by attaching to the capillary vascular endothelium (Losos and Ikede, 1973; Bungener and Muller, 1976; Banks, 1978). Consequently the concentration of these organisms may be 5 to 10 times greater in the capillary beds especially of the kidney and spleen than in the larger vessels, such as the jugular vein.

Ecology of bovine trypanosomiasis in Nigeria

The susceptibility of domestic animals, including Zebu cattle, to trypanosomiasis, contrasts strongly with the resistance to the disease observed in wild life and the dwarf-breeds of cattle (Leeflang, 1978). According to this author, trypanosomiasis exists fundamentally in a sylvatic transmission cycle in which man and livestock are not normally parasitized. This sylvatic cycle includes tse-tse flies, woody vegetation and game (Maclennan, 1963). It is only when man and his domestic animals stray into this sylvatic cycle or when infected tse-tse fly strays out of it that domestic trypanosomiasis results.

Several workers (Leeflang, 1978) have shown that bovine trypanosomiasis in Nigeria is most significant in the Sudan and Guinea vegetation zones. The coastal zones cannot be used for raising livestock because of the thick forest vegetation ubiquitously infested with tsetse fly and the lack of adequate pasture land. In the Sahel zone, which is an important livestock breeding area, tsetse flies are absent. Livestock production in this zone is therefore only limited by lack of water and inadequate grazing areas during the rather prolonged dry season which is characteristic of this zone. In the Sudan zone, tsetse flies especially

Glossina morsitans and G. tachinoides are present, mainly in woody areas and along river and flood plains. This zone provides excellent grazing area during the rainy season although there is more widespread savannah tsetse than during the dry season. In the dry season, there is a gradual depletion of available grass and water and cattle are therefore forced to graze and water in the fadama area where tsetse flies are abundant. The Guinea vegetation zone is ideal for grazing. There is usually abundant forage and water, except for a short period towards the end of the dry season when grazing is restricted to the river plains. Unfortunately, this zone is also ideal for several species of tsetse flies (Leeflang, 1978).

The above state of affair has forced the Fulani cattle owners to adopt the annual transhumance pattern of life (Asuamah, 1977; Leeflang, 1978). Essentially, the pattern followed is something like this. In the wet season, 76 percent of all the Nigerian cattle are located in the Sahel and Sudan vegetation zones. (Maclennan, 1970). Most of these areas are tsetse fly free. The remaining cattle are found in areas around Jos, Mambilla Plateau and some selected southern areas where the risk of tsetse fly is minimal. During the dry season, most of the cattle in the Sahel and Sudan vegetation have to migrate to the Guinea zones where

grazing and water are abundant and where cattle are able to withstand the tsetse fly challenges. However, these animals are consequently exposed to the danger of treking through the heavily infested savanna tsetse fly belts. After the first few rains, the cattle are then rushed back to their traditional breeding areas of the Sahel and Sudan vegetation zones where grazing and water have now become available.

Trypanosomiasis and economics of cattle production

The devastating effects of trypanosomes in the tropical livestock industry is so well known that according to Urquhart (1980), it has become customary to preface talks on animal trypanosomiasis by emphasizing the increase in livestock number which would be possible if trypanosomiasis was controlled. The importance of trypanosomiasis in cattle production particularly in Nigeria can therefore never be over-emphasized.

The interim report of a study conducted by the World Bank and Nigerian Consultants for the Federal Ministry of Agriculture and the National Committee on Green Revolution (1981) indicated that Nigeria cannot be self sufficient in beef supply till 1986. Even then the Committee recognised that Nigeria has to work hard to meet this target. Pullan and Grindle (1980) found

that the average liveweight of cows and steers in Jos Plateau were 250 kg and 350 kg respectively. This is probably true of their average respective weights throughout the country except for the exotic and dwarf breeds. The killing out percentage (KOP) is reported to be 49% (Green Revolution Report, 1981) with an average carcas weight of 125 kg. This is rather low compared to the over 60% in most temperate countries using exotic breeds. However, the reason for the low K.O.P. has been attributed to the fact that about 50% of the cattle sent to slaughter are for reasons of sickness or unthriftiness (ILCA, 1980). Although trypanosomes are not the only cause of sickness and unthriftiness among Nigerian cattle population, it may however be identified as a major factor in the low KOP because bovine trypanosomiasis is a chronic wasting disease. The Nigerian Federal Government has a policy to eradicate the tsetse flies by the use of insecticides. Remarkable success is said to have been achieved since the campaign began in 1955 (Putt et al., 1976. So far, it is reported that 19640 $km^2$ of land have been cleared in the North (94% by ground spraying and 6% by helicopters). The estimated cost is put at ₦357/$km^2$ for ground application and ₦400/$km^2$ for helicopter application. This gives ₦44.9 million for ground application and ₦4.7 million for helicopter application and a total of ₦49,587,072.00. This colossal sum from

our meagre resources could have been used for better developmental purposes. Thus the economics of tsetse fly and trypanosome in cattle and other livestock production in this country cannot be concluded without mentioning the large losses in the national revenue due to the infection. The large investment in the spraying programme does not take into account the devastating effect of the insecticide on the environment and consequently on the human health and the social problems that have been created. However, high financial benefits are expected to accrue to the livestock industry due to the tsetse eradication in the cleared areas.

Clinical trypanosomiasis in Nigerian livestock can be effectively controlled by the use of drug combinations - Novidium, Ethidium, and Berenil in high risk areas in order to prevent clinical outbreaks of trypanosomiasis. At present there are no data to show how much is spent annually on therapy though it stands to reason that the amount must be large, and must constitute an important source of economic drain to the cattle industry.

Losses from death, sickness and unthriftiness have not been calculated for the Nigerian livestock. It is postulated that this must be colossal when combined with the wastage of good grazing lands that are tsetse

infested; areas that are potentially capable of supporting livestock, one is forced to look at the figures quoted by Jasiorowski (1972) of extra 120 million cattle for Africa and by Griffin (1978) of extra 150 million sheep and extra 250 million goats for Africa if trypanosomiasis was controlled in the seven million square kilometers of tsetse infested Africa.

## TRYPANOSOMES AND BOVINE ABORTION

For years, settled Fulani cattlemen have observed that the Federal Government's sponsored tsetse fly eradication campaign and the use of adequate chemotherapy has greatly reduced the abortion rate in their herds. Several other workers have associated trypanosomes with abortion particularly in livestock. A comprehensive review of this has been published (Ogwu and Nuru, 1981). Furthermore, Murray et al (1981), in East Africa have observed that early and late pregnancy abortion occurs in Zebu cattle following natural challenge with trypanosomes. Ige and Amodu (1975) have observed infertility in cattle infected with T. vivax and T. congolense. These workers have also attributed long calving intervals in the N'dama to trypanosomiasis infection. In order to understand the mechanism of trypanosomal abortion, it is essential that we look at the possibilities of transplacental transmission of trypanosomes in animals and man.

## TRANSPLACENTAL TRANSMISSION OF TRYPANOSOMES IN ANIMAL AND MAN

Transplacental transmission of trypanosomes is the passage of the maternal infection across the placenta itself. The consequence of such fetal infection like any other fetal infection may include; fetal death and/or abortion; abortion of a live fetus; fetal malformation or dysplasia; persistent infection; subclinical infection of no serious consequence (Usburn and Kennedy, 1981) and premature delivery.

### RATS

In rats, Shaw and Dusanic (1973) have shown that Trypanosoma lewisi is capable of terminating pregnancy. This, however, depends on the time of gestation when the dam is infected. The fate of the fetuses was also shown to depend on the time of maternal infection.

Rats infected early in the first week resorbed their fetuses successfully, while those infected later in the week had greater difficulty in resorbing their fetuses. The fetuses of these dams infected at mid-term in gestation developed normally until the time of parturition, when the majority of the pregnant animals died without giving birth to their young. Animals infected during the last week of pregnancy gave birth

to litters of normal animals. In the work of Van de Branden (1935) normal fetuses were obtained from rats infected with Trypanosoma brucei. Although it was not possible to detect T. brucei microscopically in the blood of the fetuses, infection of a rat did occur, however, when it was inoculated with the fetal blood. This possibly explains why Wittebolle (1937) was unable to demonstrate the presence of T gambiense in the fetal blood of an infected rat. The presence of T. lewisi in the fetuses of infected mice was demonstrated by Minning (1936) by the development of immunity against T. lewisi by these fetuses Morenas and Roman (1937) were able to demonstrate the presence of transplacental transmission of T. brucei in rats, but they were of the opinion that this mode of transmission must be very rare.

Although most workers seem to believe that transplacental transmission of trypanosomal species does occur in rats, Gonzalez (1942) and Gillet and Herman (1976) demonstrated the absence of transplacertal passage of T. equiperdum in mice. Gonzalez (1942) showed that T. equiperdum was incapable of transplacental migration even when highly virulent strains were used.

## GUINEA PIGS

In experimentally infected guinea pigs, Kraneveld and Mansjoer (1954) were able to demonstrate the transplacental transmission of T. evansi. Although Biocca (1942) was able to demonstrate that whereas the blood of fetal guinea pigs from a dam infected with T. brucei was infective for other guinea pigs, no trypanosomes were microscopically observable in the blood of these fetuses. This observation is similar to that reported in rats and mice (Van den Branden, 1935; Wittebolle, 1937). In his work, Oleg (1941) was unable to demonstrate transplacental transmission of T. brucei in guinea pigs with a normal placenta. However, he was able to demonstrate it when the placenta was damaged by pricking or under conditions of oxygen deprivation; hence it was his opinion that the normal placenta of the guinea pig was a good barrier to trypanosomes and that the fetuses of guinea pigs can be infected only when the placenta is damaged.

## RUMINANTS

Several cases of possible transplacental transmission of trypanosomes in ruminants and hence subsequent abortions have been demonstrated (Horny, 1921; Pigneur, 1932; Dikmans et al., 1957; Lundholm et al., 1958; Lamy & Bouley, 1967; Grünert & Andressen, 1970;

Woo & Limebeer, 1971; Paikne & Dhake, 1972; Ikede & Losos, 1972; Moulton & Kraus, 1972; Isoun & Anosa, 1974; and Nuru, 1974). In the buffalo, Paikne and Dhake (1972) reported the abortion of a fully developed calf which was positive for T. evansi. The abortion was attributed to T. evansi, and subinoculation of the fetal blood from the calf to rabbits resulted in the deaths of the rabbits. Ikede & Losos (1972) inoculated sheep intravenously with T. vivax and reported the birth of a weak lamb 33 days later that was positive for T. vivax. Although Isoun and Anosa (1974) did not report a similar finding, they were able to demonstrate the presence of T. vivax in amniotic fluid of a pregnant sheep that had been infected experimentally.

In cows, Hornby (1921) suggested that T. vivax is capable of causing abortion of weak calves. Although he did not speculate on the mechanism involved, it is now believed that trypanosomes are capable of transplacental migration in pregnant bovine females. There is other evidence for this, notably from the work of Dikmans et al., (1957) who found T. theileri in the stomach contents of an aborted calf. Lundholm et al. (1959) confirmed the suggestion of Dikmans et al., (1957), that transplacental transmission of trypanosomes in cattle was possible by their demonstration of

T. theileri as a contaminant of cell cultures of bovine fetal kidney cells. In 1967, Lamy & Bouley reported heavy trypanosomal parasitemia in a newly born calf. Pigneur (1932) detected T. vivax in the blood of a prematurely born calf of an infected cow; inoculation of blood from the calf into a pregnant goat resulted in the birth of an infected premature kid. Another case demonstrative of transplacental transmission of trypanosomes in the bovine female was reported by Moulton & Krauss (1972) who found T. theileri as a contaminant of cell cultures of bovine fetal spleen. The trypanosomes in this were initially intracellular but eventually became extracellular. Woo & Limebeer (1971) reported the presence of trypanosomes in cell cultures from the spleen and kidneys of an 8-month-old calf fetus. The trypanosomes were first noticed in the spleen cultures. They were dividing and were all of the trypamastigote forms. The authors also found that there was a higher indicence of trypanosomes in spleen cultures than in kidney culture and attributed this to the larger number of trypanosomes in the sinusoids of the spleen than in the capillaries of the kidney.

Contrary evidence on the transplacental transmission of trypanosomes has come from cultural surveys of cattle in Kenya (Reid et al., 1967) and Nigeria (Gray and Nixon, 1967) which showed that infection rates were

lower in young than in older animals, and did not reveal any congenital infections in calves from infected cows. These findings indicate that the transplacental route may be an abnormal method of transmission.

More recently, Nuru (1974) conducted four experiments on the effect of T. vivax on pregnant cows following natural infection; the effect of T. vivax on susceptible pregnant cows challenged intravenously; the effect of T. vivax on pregnant cows following intra-amniotic challenge; the intravenous challenge of pregnant cows with T. vivax and subsequent termination of pregnancy. It was his opinion that T. vivax does not directly cause abortion per se. It is interesting to note that T. vivax, although not detected directly in the blood of fetuses from the last experiment, was detected in a susceptible cow that had been inoculated with the blood from the fetuses. This work, although suggestive, was inconclusive.

Perhaps the earliest reported evidence of transplacental transmission of trypanosomes in domestic animals was that of Sergent and Lheritier (1919). In their pioneering work, the authors inoculated two camels with T. berberum. Both camels aborted dead fetuses almost at term. In neither case it was not possible to demonstrate any trypanosomes in the spleen of the aborted fetuses. However, filtrates of crushed spleen

in both cases produced infections in the inoculated dogs. This was the first documented evidence of a trypanosome species being able to cross the placental barrier.

## HORSES

There is apparently little literature on the possibility of transplacental transmission of trypanosomes in horses. There is however, one report from Kraneweld & Mansjoer (1954). Using T. evansi, they demonstrated that inoculation of this organism into pregnant mares resulted in fetal deaths and their subsequent mummification.

## M A N

Human fetuses too are reported to be infected from their mothers. Bassett-Smith (1919) had considered transplacental transmission of trypanosomes a possibility. His suggestion was strengthened by Kellersberger (1925) who found a large number of trypanosomes in the blood of a 3-week-old baby. Perhaps the first confirmed case report of the transplacental transmission of trypanosomes in man was that of Darre et al., (1937). The child was born in France in 1934, but the mother had been infected while pregnant in some parts of central

and west Africa. The child was under weight (2.4 kg) and had an abnormally large head. After several months of test, trypanosomes were later discovered in the cerebrospinal fluid. Anti-trypanosome drug therapy gave good results and the child lived. A more recent human case of the transplacental transmission of trypanosomes was reported by Olowe (1975) from Lagos, Nigeria. Here a ventricular tap was performed on the baby and the cerebrospinal fluid was found to be swarming with trypanosomes even though no trypanosomes were seen in the blood. The mother was positive for trypanosomes.

## MECHANISMS OF TRANSPLACENTAL TRANSMISSION OF TRYPANOSOMES

From the above, there is some evidence for the possible transplacental transmission of trypanosomes in the rat, guinea pig, sheep, cow, buffalo, camel, mare and man. However, some of these species have different types of placenta; equines and ruminants have the epitheliochorial type of placenta, man, rabbits and guinea pigs have the haemochorial type, while rats and mice have the endotheliochorial types of placenta (Bjorkman, 1954, 1964, 1965ab). For the fetus to be infected from the maternal circulation, trypanosomes must be capable or have a means of passing

through tissue barriers. Trypanosomes are known to poss_ss biologically active products (Tizard, 1978b). Of interest here are enzymes: proteases, phospholipases and a vascular-permeability-increasing factor .

Trypanosomes possess both acid phosphatase and cathepsin D (Venkatesan et al., 1977). These enzymes together with other proteases may act on a number of biologically significant substrates including plasminogen and fibrinogen (Borcham & Facer, 1973). Phospholipases are the second group of enzymes which trypanosomes are known to posses (Tizard et al., 1977, 1978): these are capable of destroying cell membrane phospholipids. As T. vivax and T. congolense lodge preferentially within the microcirculation by attaching to the capillary vascular endothelium (Losos & Ikede, 1973; Bungener & Muller, 1976; Banks, 1978), it is easy to see how trypanosomiasis in pregnant animal may result in placental damage. It must, however, be emphasized that the enzymes are released only on lysis of the trypanosomes. The presence of neuraminidase has been detected by Esievo (1980). This enzyme is known to suppress pregnancy in mice (Gasic and Gasic, 1970) as well as altering the antigenic make-up of trophoblasts thus resulting in abortion and infertility (Muller and Saathoff, 1972). In addition to enzymes T. brucei has been shown to posses a vascular permeability

increasing factor (Goodwin & Guy, 1973), though the precise nature of this factor remains unknown.

Trypanosomes have been identified in the experimentally infected dams, and transplacental transmission appears to be the only plausible mode of infection of those fetuses. However, no one has ever produced a satisfactory explanation as to the mechanism by which this organism is able to cross the placenta. Nuru (1974) in his thesis stated that the method by which trypanosomes cause abortion is not known. One mechanism often postulated is that there may be damage to the placenta (Njoku, 1980, personal communication). It is perhaps important to state here that no one so far has been able to demonstrate placental damage in animal trypanosomiasis.

## IMMUNOGLOBULIN LLVELS IN TRYRPANOSOMIASIS

In mammals, immune response to infection is characterised by the sequential synthesis of different classes of immunoglobulins in which the initial immunoglobulin M (IgM) synthesis is replaced by immunoglobunlin G (IgG) synthesis (Baner & Stavitsky, 1961 and Barrett, 1974). In trypanosomiasis, this sequence is modified and the infection is characterised by the prolonged production of IgM antibodies (Masseyeff and

Lamy, 1966, Lucking, 1972, 1973, 1976). This disease is not only characteristic by this prolonged production of IgM but the IgM levels may be greatly elevated (Klein et al., 1967; Houba et al., 1969; Rees, 1969; Seed, 1969; Baker & Taylor, 1971; Luckins, 1972, 1976; Kobayashi et al., 1976) rising on some occasions, 20 times the pre-infection values (Kobayashi et al., 1976). In contrast IgG levels tend to remain constant at values close to normal (Kobayashi & Tizard, 1976) though values 2-4 times the preinfection levels have been reported (Rees, 1969, Luckins, 1972).

This prolonged synthesis of IgM, though not pathognomonic, has been used as a basis for the diagnosis of trypanosomiasis. In West Africa, the importance of the estimation of serum IgM as a screening test for the diagnosis of T. gambiense has been demonstrated by Mattern et al., (1961) and Mattern (1962, 1964, 1968). Other workers have also validated this screening test including Binz et al. (1968); Goidl and Marsden (1974) and Lumsden (1966).

This tremendous production of IgM seems to be directed against a number of antigens. Some of these are specifically antitrypanosomes (Seed et al., 1969) while others may be heterophils (Houba et al., 1969; Mackenzie, 1973) or directed against autoantigens (Mansefield and Kreir, 1972; Mackenzie et al., 1973).

Few observations have been made on the effect of trypanosomiasis on serum immunoglobulin levels in cattle (Desowitz, 1959, Luckins, 1972, 1976).

Using Zebu cattle from tse-tse free areas and allowing them natural challenge, Luckins (1972) found that there was a significant increase in IgM and IgG levels. Increases of 2-9 times preinfection levels of IgM were reported 2 weeks after the cattle were introduced into the enzootic area and there persisted for several months. IgG levels were reported to show only a 2-fold increase during the period.

In this later work, using experimental T. congolense and T. vivax infection, Luckins (1976) found that increases in IgM levels occurred within 14 days of infection coinciding with the first appearance of the trypanosomes in peripheral blood. Increases of 2-10 times were recorded. IgG levels showed only little increase. The animal that died of the infection showed only an initial increase in IgM levels but subsequently IgM and IgG levels decline until the animal died. The levels of IgM were also reported to have declined to approximately the pre-infection levels within 25 days following diminazene aceturate treatment.

# PART II

## EFFECT OF EXPERIMENTAL T. VIVAX INFECTION ON FERTILITY OF HEIFERS

# INTRODUCTION

Although sterility and abortion are among the symptoms of human sleeping sickness (Apted, 1970), the situation in animal trypanosomiasis is not quite clear (Ikede, 1975). In humans, the reproductive problems associated with sleeping sickness are attributed partially to endocrine dysfunction, especially hypopituitarism (Apted, 1970). In cattle there is sufficient evidence of abortions caused by trypanosomes (Krampitz, 1970, Nuru, 1974 and Ogwu, 1982, unpublished data). The mechanism of this abortion has not been clearly worked out but may have to do with transplacental transmission (Ogwu and Nuru, 1981), fetal pathology (Ogwu, 1982), and placental damage (Njoku personal communications).

The role of trypanosomes in the fertility of cattle in endemic areas has not been fully investigated. In one study, Ige and Amodu (1975) found that there was irregular estrus in heifers infected with T. congolense and infertility occurred. In one heifer, there was irregular estrus 9 months post infection, then it had a healthy calf and never returned to estrus for up to 8 months. Using T. brucei, these workers found no signs of heat by infected animals until 8 - 9 months. With T. vivax infection no signs of estrus were observed

in 3½ months after the disappearance of the parasitemia. Infertility and long calving intervals observed in endemic areas of bovine trypanosomiasis were attributed to the disease.

Although no reasons were given by Ige and Amodu (1975) for the observed infertility, this may be due to the hypophysial lesions in association with animal trypanosomiasis(Losos and Ikede, 1970; McCully and Nietz, 1971, Ikede and Losos, 1975).

This paper reports the findings of an investigation set up to examine the effect of experimental infection of T. vivax, Y56, on the fertility of bovine heifers.

# MATERIALS AND METHODS

## Experimental Animals

A group of fifteen 3-year-old heifers were acquired from the Kano State Government-owned LIBC farm at Gumel, Nigeria, a tsetse fly free area. The heifers were selected after careful examination including that of the ovaries and genitalia. The animals were transported in a truck to the Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria and housed in the research pen of the Faculty.

Two days after their arrival here, they were dipped using Pfizona (Pfizer Products Ltd; Ikeja, Nigeria) and dewormed with Thiabendazol (Thibenzol; Merck Sharp and Dohme) and Ranide(R) (Merck Sharp and Dohme) 2 weeks apart. The animals were screened for brucellosis. In addition, their blood was evaluated for baseline hematological data and for the presence of blood parasites, which if present were treated with Imizol (The Wellcome Foundation Ltd; London) and/or Maxitet (Beecham, Animal Health, Brentford, England). The animals were fed grass hay supplemented with cotton seed cake and wheat offals for 6 months before the experiments and during the experimental period. They were given mineralised salt lick (Pfizer Products Ltd; Ikeja, Nigeria) and water ad libitum. At the end of the

experiment infected animals were treated with Novidium chloride (Major and Co. (Nig.) Ltd., Kano, Nigeria).

Trypanosomes

The Y58 strain of T. vivax isolated from a natural infection in N'dama cow (Leeflang et al., 1976) was used.

Stabilate used was preserved in 5% DMSO and stored in liquid nitrogen. Prior to use in the present experiment, the stabilate was first inoculated into sheep which subsequently developed clinical trypanosomiasis. Blood from this sheep was then used for the experiment.

Experimental Design

Six heifers were selected from the group of 15. Four of the selected heifers had corpora lutea and were on days 10, 11, 12 and 12 respectively of their individual estrous cycles. The other 2 heifers serving as controls were on days 6 and 8 respectively of their estrous cycles. Because of prior knowledge of the estrous cycle length of these heifers and the prepatent period of the strain of T. vivax Y58, the experimental heifers were inoculated intravenously with approximately $1.5 \times 10^6$ T. vivax on day 16 for

the first 2 and day 14 for the last 2. The controls were not inoculated. The 6 heifers were kept in a pen with the proven bull. The animals were observed closely until they were seen to be bred. After the breeding, the bull and the 2 control heifers were removed and kept in separate pens. Following inoculation, the animals (including the controls) were bled daily for the first 3 weeks and there after thrice a week. Serum samples were collected once a week. Blood collections were via the jugular venipuncture into vacutainer tubes with ethylene diamene-tetraacetic acid (EDTA) (1mg/ml blood) as anticoagulant. Samples for sera collection were collected in plain vacutainer tubes without anticoagulant. Such samples were allowed to coagulate at room temperature for 4 hours and centrifuged at 1000 RPM for 10 minutes at $4^{o}C$. The sera were separated out into labelled serum vials and stored at - $20^{o}C$ until assayed. On each occasion, rectal temperatures were taken before bleeding.

Haematocrit Centrifuge Test for Trypanosomes

A microhaematocrit capillary tube containing approximately 70 ul of blood was centrifuged for 5 minutes just as for measurement of PCV. The tubes were then placed on a clean glass slide. A drop of immersion oil was put on top of the capillary tubes

with the oil filling the spaces between the capillary tubes. The buffy coat plasma junction was then examined under the microscope using the oil immersion objectives as described by Woo (1969). The degree of parasitemia was classified as follows:

\+ = occasional trypanosomes seen
++ = 1 to 2 trypanosomes seen
+++ = 3 to20 trypanosomes seen
++++ = over 20 trypanosomes seen

Thin Blood Films for Trypanosome Identification

Clean, grease-free microscope slides were used. About 5 microlitres of blood were placed at one end of the slide and a thin smear made with the aid of another microscope slide. The slides were quickly air dried and fixed for 3 minutes in methanol. After this the slides were stained in 10% Giemsa, dried and about 100 fields scanned under oil immersion (x 790).

Packed Cell Volume (PCV)

In order to estimate the degree of anemia the packed cell volume (PCV) was determined by the microhaematocrit method using the international microhaematocrit centrifuge.

Reproductive and Pregnancy Examination

During the 6 months acclimatization period, reproductive evaluation of the heifers was carried out. To do this the heifers were examined rectally after proper restraint in a chute on the day of estrus and weekly thereafter. To achieve this, an obstetrical glove is worn and lubricated with soap. The hand is introduced into the rectum and the ovaries and genitalia palpated as described by Zemjanis (1970). Pregnancy examination was carried out during the selection at the LIBC farm at Gumel, on arrival at the Faculty, 40 days post breeding and two weeks thereafter.

# RESULTS

All the heifers exhibited estrus between days 20 and 22. Not much external signs were shown. The heifers first started to mount other heifers and then stood to be mounted. When the bull was introduced the heifers did not stand to be mounted until about 6 hours after the onset of estrus. In all, the length of estrus was about 16 hours (± 2.5) and varied slightly with the different heifers. In addition, estrus in these heifers was marked by restlessness and the vulva in all the animals was slightly edematous and relaxed. The mucosa of the vagina was pink and congested. Strings of clear mucus were shown by all the animals and in these heifers, the mucus marked the peak of sexual receptivity. Thereafter the heifers did not accept the bull again.

Rectal examination at the time of estrus showed a highly tonic genitalia and a matured Graafian follicle.

Although the bull attempted to mount the heifers early during estrus, intromission was not allowed as the heifers tended to "jump" and ward off the bull. The bull however continued to follow and lick the vulva of the heifer on heat until it allowed him to breed her. All the heifers were bred once.

Following breeding, the heifers showed signs of pain. The tails were elevated for sometime. There was tenesumus and attempts at frequent micturation. No metestrous haemorrhage was observed in the animals.

Clinical Signs and Parasitemia

All the infected heifers developed trypanosomiasis. After the first 4 days, the heifers became increasingly lethargic. Their hair coats become dull and their mucous membranes pale in color following the onset of anemia. There was a gradual loss of condition. Lacrimation was evident in all the infected heifers and the superficial lymph nodes were noticeably enlarged. Despite these signs, the animals continued to eat and had good appetite.

From the third day postinfection, the heifer started exhibiting increases in rectal temperature rising on occasions to 40°C and fluctuating. The rectal temperature in the infected animals was statistically higher than those in the non-infected controls ($P<0.001$). The temperature returned to normal following the disappearance of the parasites from peripheral blood. The fluctuations in rectal temperature, parasitemia and packed cell volume are shown in tables 1 & 2 and summarised in fugure 1.

Trypanosomes were detected in the peripheral circulation of the heifers on day 4 pi in one heifer and on the fifth day in the remaining 3. Thereafter, there were fluctuations in the degree of parasitemia. The peak of the parasitemia occurred between the 8th and 12th day pi and continued to fluctuate thereafter. Parasites were still detectable in these animals after three months pi.

The packed cell volume (PCV) decreased from the 6th day and continued to decrease gradually. The PCV values in the infected heifers were lowest between the 20th and 50th day post infection. These values were statistically different from those of the controls ($P<0.001$). In most of the infected animals the PCV started to rise after about 100 days post infection. The rise was gradual and never reached the preinfection levels.

It was observed that all the infected heifers developed a chronic type of trypanosomiasis. This is probably due to the strain of Trypanosoma vivax (Y58) used. No acute trypanosomiasis was recorded in any of the infected heifers.

Effect on Fertility

All the 6 animals, 4 infected and 2 controls, were bred within one week. Rectal examination of the ovaries one week later showed fully developed corpora lutea in all the heifers and no significant difference was detected in the sizes of the corpora lutea.

Rectal examination of the heifers 40 days post breeding showed that while all the 4 infected heifers were not pregnant, the 2 controls were pregnant. In addition no functional structures, follicles or corpora lutea, were palpable on the ovaries of the heifers. The heifers did not return to estrus for over 5 months.

# DISCUSSION

The mouse infective strain of Trypanosoma vivax (Y58) used in this experiment caused clinical chronic trypanosomiasis in all the heifers. This result showed that the Zebu cattle were able to withstand a single infection of T. vivax. In the field, however, continuous challenge occurs. This, coupled with poor nutrition and tick borne infections are probably responsible for the fatality seen. In this experiment these 2 factors were eliminated and no fatality was recorded.

The results also showed that T. vivax infection was characterised by high fluctuating parasitemia and significantly high rectal temperatures. The animals were bred at the onset of these 2 factors and no pregnancy resulted. Since the heifers ovulated and were bred, one can infer that infection with T. vivax resulted in the infertility.

There are ways by which T. vivax infection could have resulted in this conception failure. The first is the rise in body temperature of the heifers. It is well known that hyperthermia causes morphological abnormalities of eggs e.g. shrunken cytoplasm, ruptured vitelline membranes or ruptured zona pellucida (Hafez, 1968). Dunlap and Vincent (1971) found that thermal stress after mating had disastrous effects on beef

heifers exposed to 32°C for 72 hours immediately after insemination. In dairy cows, Wiersma and Stott (1966, 1969) found that the first 4-6 days after service were most critical. Although these studies were done with ambient temperature, infections producing high body temperatures are likely to produce the same effect if the high body temperatures coincide with the time of mating. In this study breeding was done in all the experimental animals at the onset of the hyperthemia. Thus it is possible that the high body temperatures resulting from T. vivax infection was in part responsible for the conception failure/embryonic death.

Thermal stress has been known to affect the uterine environment. Research in embryo transfer has provided adequate information on the importance of the state of the uterine environment for the viability and development of the embryo (Rowson et al., 1972). High ambient temperatures in recipients were known to affect the uterine environment. Thus it is known that thermal stress acts directly on the milieu interieur rather than on the embryo itself (Hafez, 1968). It is likely that high body temperatures produce the same effect. Recent study by Zakari et al., (1981) has shown that elevated rectal temperature was detrimental to conception.

Since high temperatures affect the uterine environment, the survival of the ejaculated sperm cells may be affected apart from the direct effect of high body temperature in the female on the cells. Thus it is likely that the high body temperature in the infected female produced unfavourable environment for the sperm cells ejaculated during mating. In addition the high body temperature of the heifers may have affected the cells directly thus resulting in conception failure. In sheep, Ulberg and Burfening (1967) reported a correlation between body temperature near the time of mating and subsequent embryo survival. Similar results have been reported by Ulberg and Sheean (1973).

The second possible way by which T. vivax may have caused the conception failure is by the production of neuraminidese (= sialidase EC 3.2.1.18). The strain of T. vivax used (i.e. Y58) has been shown to produce neuraminidase (Esievo, 1980).

Other workers (Muller and Saathoff (1972) have shown that organisms producing neuraminidase are capable of causing abortion and infertility by altering the antigenic make-up of trophoblasts. In another study, Gasic and Gasic (1970) demonstrated total suppression of pregnancy in mice by post-coital administration of neuraminidase, which interferes with implantation.

Since no conception occurred in any of the infected animals in this study, one might infer that neuraminidase may be responsible for the conception failure or at least one of the factors responsible for the failure.

The third possibility to account for the conception failure/embyonic death, is the direct effect of T. vivax itself on the pregnant uterus and or embryo. Since tissue reactions have been observed in bovine species infected with T. vivax (Isoun and Esuruoso, 1972) and transplacental transmission is possible in bovine trypanosomiasis (Hornby, 1921, Dikmas et al., 1957; Lundholm et al., 1959, Lamy and Bouley, 1967; Pigneur, 1932; Nuru, 1974 and Ogwu and Nuru, 1981) it is therefore possible that there is a direct invasion of the embryo resulting in death and subsequent resorption.

The results from this study showed that experimental T. vivax infection caused the suspension of cyclic activities in the infected heifers. Similar observation has been made by Ige and Amodu (1975). These workers observed that 3 females infected with T. vivax did not come into heat until 3½ months after the disappearance of parasitemia. Although no evidence is available from this present work to account for this anestrus, other workers (Wiersma and Stott, 1966 and

1969) have shown that increased body temperatures depressed plasma progesterone and corticosteroid levels. In addition, protracted anemia resulting from trypanosome infection is known to affect pituitary function (Sannusi, personal communication). Of importance here, however, is the finding of Ikede and Losos (1975) and those of Morrison et al., (1981).that hypophysial lesions do occur in sheep and dogs respectively infected with T. brucei. Although no such findings have been reported in T. vivax infection (probably because there is less research done with the organism)it is possible that hypophysial lesions could occur.

Estrous activity is controlled by an intricate balance between the gonadotropin releasing hormones (GnRH, the gonadotropins Follicle Stimuting hormone, FSH, and Luteining hormone, LH, and sex steroids (estrogen and progesterone). The hypophysis, particularly the adenophypophysis, is perhaps one of the most important organs in this carefully integrated hormonal process. Therefore, it is easy to see why an infectious agent capable of producing hypophysial pathology can result in cesation of cyclic activities and therefore result in anestrous. More work is definitely required to establish the hypophysial pathology in cattle infected with T. vivax, and to study the reproductive hormone levels in heifers infected with the organism.

# S U M M A R Y

A group of four experimental heifers and two controls were used to study the effect of Trypanosoma vivax infection on fertility. The group (3-year-old Zebu heifers) was intravenously inoculated with T. vivax - strain Y58 on day 14 and 16 of their estrous cycle. The two control heifers in the same phase of estrus were uninfected. The animals together with the controls exhibited estrus and were bred in the research pens with a proven bull. The infected animals were bred at the first wave of parasitemia corresponding with the onset of pyrexia which characterised the infection. Other clinical signs of trypanosomiasis were not apparent at the time of breeding. Later, clinical signs of the infection appeared which among others included intermittent fever with temperature rising sometimes to 40°C, anemia, progressive cachexia, lacrimation and lymphatic enlargement. The four experimental heifers were found open while the two controls were pregnant when examined rectally 40 days later. In addition, the heifers did not come back to estrus during the experimental period of four months. It is concluded that bovine trypanosomiasis, among other things, may be responsible for the high infertility rate observed in cattle kept in endemic areas.

Table 1: Means and standard deviations of PCV and rectal temperature in four non-pregnant heifers experimentally infected with T. vivax strain Y58

| Days of Experiment | PCV % | Temperature °C |
|---|---|---|
| -8 | 42.6 ± 1.84 | 38.5 ± 0.08 |
| -3 | 42.0 ± 2.44 | 38.5 ± 0.07 |
| 0 | 42.0 ± 2.44 | 38.4 ± 0.2 |
| 1 | 42.0 ± 2.27 | 38.4 ± 0.07 |
| 2 | 42.1 ± 2.32 | 38.4 ± 0.05 |
| 3 | 34.4 ± 16.33 | 38.6 ± 0.05 |
| 4 | 42.0 ± 2.16 | 38.9 ± 0.3 |
| 5 | 41.5 ± 2.64 | 39.0 ± 0.4 |
| 6 | 41.5 ± 1.95 | 39.1 ± 0.4 |
| 7 | 39.7 ± 2.06 | 39.4 ± 0.5 |
| 8 | 39.1 ± 2.01 | 39.8 ± 0.2 |
| 9 | 38.7 ± 1.50 | 39.9 ± 0.05 |
| 10 | 37.7 ± 1.50 | 39.9 ± 0.1 |
| 11 | 36.3 ± 1.79 | 39.9 ± 0.05 |
| 12 | 36.5 ± 1.41 | 40.3 ± 0.3 |
| 13 | 35.5 ± 2.27 | 40.1 ± 0.3 |
| 14 | 35.5 ± 2.04 | 40.1 ± 0.3 |
| 15 | 31.7 ± 2.87 | 40.1 ± 0.3 |
| 16 | 31.7 ± 2.87 | 40.0 ± 0.3 |
| 17 | 31.0 ± 2.70 | 39.9 ± 0.1 |
| 18 | 28.7 ± 1.50 | 39.8 |
| 21 | 24.3 ± 2.86 | 39.8 ± 0.2 |
| 25 | 23.7 ± 1.70 | 39.6 ± 0.4 |
| 28 | 22.6 ± 2.68 | 39.8 ± 0.5 |
| 31 | 21.1 ± 2.59 | 39.7 ± 0.2 |
| 36 | 20.3 ± 3.14 | 39.1 ± 0.6 |
| 39 | 18.1 ± 0.25 | 38.9 ± 0.06 |
| 42 | 17.7 ± 1.19 | 39.0 ± 0.3 |
| 44 | 16.0 ± 2.16 | 38.8 ± 0.2 |
| 49 | 17.1 ± 2.56 | 39.4 ± 0.9 |
| 51 | 17.3 ± 2.91 | 39.0 ± 0.6 |
| 53 | 18.5 ± 1.91 | 38.9 ± 0.6 |
| 58 | 18.6 ± 1.25 | 38.6 ± 1.2 |
| 60 | 18.8 ± 1.18 | 38.8 ± 0.2 |
| 63 | 20.1 ± 1.65 | 38.8 ± 0.4 |
| 65 | 20.7 ± 1.65 | 39.1 ± 0.2 |
| 70 | 21.5 ± 2.54 | 39.0 ± 0.6 |
| 72 | 22.5 ± 2.38 | 39.0 ± 0.6 |
| 77 | 23.3 ± 2.49 | 39.3 ± 0.6 |
| 79 | 23.7 ± 2.63 | 39.1 ± 0.4 |
| 81 | 24.7 ± 2.75 | 38.6 ± 0.2 |
| 85 | 25.1 ± 2.78 | 38.7 ± 0.3 |
| 86 | 25.1 ± 2.78 | 38.6 ± 0.1 |
| 88 | 25.6 ± 2.39 | 38.6 ± 0.2 |
| 91 | 25.7 ± 2.59 | 38.9 ± 0.7 |

| Days of Experiment | P&V % | Temperature °C |
|---|---|---|
| 93 | 25.0 ± 3.26 | 38.5 ± 0.1 |
| 95 | 25.5 ± 3.48 | 38.6 ± 0.2 |
| 98 | 23.5 ± 2.79 | 38.6 ± 0.2 |
| 100 | 26.3 ± 2.56 | 38.5 ± 0.2 |
| 102 | 26.8 ± 2.49 | 38.5 ± 0.1 |
| 109 | 26.7 ± 2.53 | 38.4 ± 0.05 |
| 115 | 27.0 ± 2.34 | 38.5 ± 0.05 |
| 122 | 27.1 ± 2.01 | 38.5 ± 0.08 |

Table 2: Means and Standard deviations of PCV and rectal temperature in two non-pregnant control heifers.

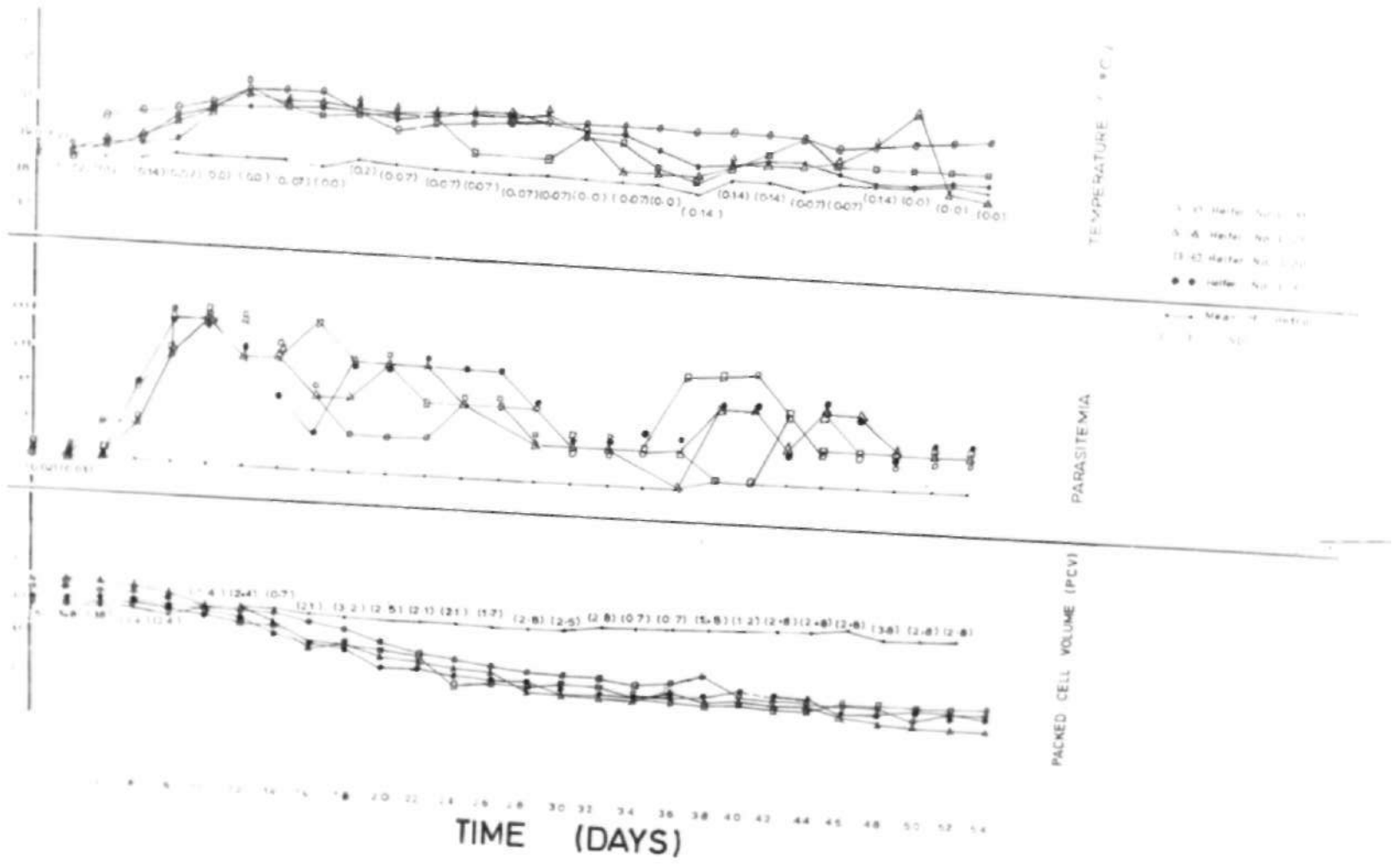
| Days of Experiment | PCV % | Temperature $^{o}C$ |
|---|---|---|
| -8 | 37.8 ± 3.18 | 38.45 ± 0.07 |
| -3 | 38.5 ± 3.53 | 38.45 ± 0.21 |
| 0 | 38.7 ± 4.59 | 38.45 ± 0.07 |
| 1 | 38.5 ± 4.24 | 38.45 ± 0.07 |
| 2 | 37.5 ± 3.53 | 38.50 ± 0.00 |
| 3 | 37.7 ± 3.88 | 38.50 ± 0.00 |
| 4 | 37.7 ± 3.88 | 38.50 ± 0.00 |
| 5 | 39.7 ± 2.47 | 38.40 ± 0.14 |
| 6 | 39.5 ± 2.12 | 38.35 ± 0.07 |
| 7 | 39.0 ± 1.41 | 38.40 ± 0.14 |
| 8 | 38.3 ± 2.47 | 38.50 ± 0.14 |
| 9 | 38.5 ± 0.70 | 38.45 ± 0.14 |
| 10 | 40.00 ± 2.12 | 38.50 ± 0.00 |
| 11 | 38.7 ± 3.12 | 38.50 ± 0.00 |
| 12 | 39.7 ± 2.47 | 38.50 ± 0.00 |
| 13 | 38.5 ± 2.12 | 38.45 ± 0.07 |
| 14 | 38.5 ± 2.12 | 38.50 ± 0.00 |
| 15 | 38.7 ± 1.76 | 38.55 ± 0.07 |
| 16 | 39.0 ± 2.82 | 38.40 ± 0.00 |
| 17 | 38.3 ± 2.47 | 38.50 ± 0.00 |
| 18 | 38.0 ± 2.82 | 38.55 ± 0.21 |
| 19 | 38.5 ± 0.70 | 38.55 ± 0.07 |
| 21 | 38.5 ± 0.70 | 38.55 ± 0 07 |
| 25 | 39.2 ± 1.76 | 38.60 ± 0.14 |
| 28 | 39.7 ± 1.76 | 38.55 ± 0.07 |
| 31 | 39.0 ± 2.82 | 38.55 ± 0.07 |
| 36 | 39.0 ± 2.82 | 38.55 ± 0.07 |
| 39 | 40.0 ± 2.82 | 38.40 ± 0.14 |
| 42 | 40.0 ± 2.82 | 38.50 ± 0.00 |
| 44 | 38.2 ± 3.88 | 38.55 ± 0.07 |
| 49 | 38.0 ± 2.82 | 38.45 ± 0.07 |
| 51 | 38.0 ± 2.82 | 38.45 ± 0.07 |
| 53 | 38.5 ± 3.53 | 38.40 ± 0.14 |
| 58 | 38.5 ± 3.53 | 38.55 ± 0.07 |
| 60 | 38.0 ± 2.82 | 38.50 ± 0.00 |
| 63 | 38.0 ± 2.82 | 38.40 ± 0.14 |
| 65 | 39.7 ± 2.47 | 38.50 ± 0.00 |
| 70 | 39.7 ± 2.47 | 38.50 ± 0.00 |
| 77 | 38.00 ± 2.82 | 38.45 ± 0.07 |
| 79 | 39.0 ± 1.41 | 38.55 ± 0.07 |

| Days of Experiment | PCV % | Temperature °C |
|---|---|---|
| 81 | 38.2 ± 2.47 | 38.40 ± 0.14 |
| 85 | 38.5 ± 3.53 | 38.45 ± 0.07 |
| 86 | 39.5 ± 2.12 | 38.40 ± 0.00 |
| 88 | 38.5 ± 2.12 | 38.50 ± 0.14 |
| 91 | 37.2 ± 2.47 | 38.45 ± 0.07 |
| 93 | 37.0 ± 2.82 | 38.50 ± 0.14 |
| 95 | 38.2 ± 3.18 | 38.50 ± 0.00 |
| 98 | 38.00 ± 2.82 | 38.60 ± 0.14 |
| 100 | 38.2 ± 2.47 | 38.50 ± 0.00 |
| 102 | 38.2 ± 2.47 | 38.45 ± 0.07 |
| 109 | 38.2 ± 2.47 | 38.45 ± 0.07 |
| 115 | 39.0 ± 2.82 | 38.45 ± 0.00 |
| 122 | 38.5 ± 4.94 | 38.55 ± 0.07 |
| 129 | 40.5 ± 0.07 | 38.50 ± 0.00 |

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Figure 1

Temperature curves, parasitemia and packed cell volume (PCV) in Zebu heifers infected with Trypanosoma vivax near time of estrus.


TEMPERATURE (°C)
PARASITEMIA
PACKED CELL VOLUME (PCV)
TIME (DAYS)

# PART III

## THE EFFECTS OF TRYPANOSOMA VIVAX ON FIRST, SECOND AND THIRD TRIMESTER PREGNANCY IN HEIFERS

# INTRODUCTION

For many years, tse-tse transmitted trypanosomisis particularly in cattle, has been the major constraint to meat production in Africa. Ten million sq. kilometres of land in Africa is said to be tse-tse fly infested, requiring about 2 - 3 billion U.S. dollars to eradicate (David-West, 1981). In Nigeria, trypanosomiasis is responsible for the nomadism and transhumance with all their attendant socio-economic and political problems (Asuamah, 1977). It is also responsible for the restriction of ruminants especially cattle to the Sahel and Northern Sudan vegetation zones where rainfall and therefore pasture is limited and rendering the lush pastures of the South unutilized for grazing. Reasons have been advanced (Tizard et al., 1978, Ogwu and Nuru, 1981) on why, inspite of the apparent technological developments in medicine, pathogenic trypanosomes continue to maintain their lethal grip on both human and domestic animals in most parts of subsaharan Africa. The pathogenesis and immunobiology of African trypanosomiasis has been the subject of many recent reviews (Ikede, 1975; Murray, 1975; Mansfield, 1978; Tizard et al., 1978).

However, inspite of the devastating effects of trypanosomes on livestock production, very little has been done regarding the role of these pathogens on

bovine reproduction. The review by Ogwu and Nuru (1981) has indicated that transplacental transmission of trypanosomes may be a possibility in animals and men. In the cow, several workers (Hornby, 1921; Dikmans et al., 1957; and Lundholm et al., 1959) have shown that trypanosomes are capable of causing abortion, premature or weak calving. Of interest here is the finding of Shaw and Dusanic (1973) that in the rat, the time of maternal infection during pregnancy affects the fate of the fetuses.

This work was carried out to show whether T. vivax, infection does indeed cause abortion, premature and or weak calving. In addition, the work was designed to ascertain at what stage of pregnancy the infection causes abortion, premature or weak calving and to determine, if possible, the mechanism by which these disturbances of pregnancy are brought about.

# MATERIALS AND METHODS

## Experimental Animals

Twenty four heifers aged 3 years were acquired from the Kano State government Livestock Investigation and Breeding Centre (LIBC) farm at Gumel and from National Animal Production Research Institute (NAPRI) farm at Shika, Nigeria. The heifers were selected after careful examination which included that of the ovaries and genitalia. The animals were transported in a truck to the Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria, Nigeria and housed in the research pen of the Faculty.

Two days after their arrival at the Faculty, they were dipped using Pfizona$^{R}$ (Pfizer Products Ltd., Ikeja, Nigeria) and dewormed with Thiabendazol$^{R}$ (Thiabenzol, Merck Sharp and Dohme, England) and Ranide$^{R}$ (Merch Sharp and Dohme, England) 2 weeks apart. The animals were screened for brucellosis and leptospirosis. In addition, their blood was evaluated for baseline hematological data and for the presence of blood parasites. The animals that were positive for piroplasms were treated with Imizol$^{R}$ (The Wellcome Foundation Ltd., London) and/or Maxitet$^{R}$ (Beecham Animal Health, Brentford, Middlesex, England). The animals were fed grass hay supplemented with cotton seed cake

and wheat offals for 6 months before the experiments and during the experimental period. They were also given mineralised salt lick (Plizer Products Ltd., Ikeja, Nigeria) and water ad libitum. At the end of the experiments, the non-pregnant infected animals were treated with Novidium chloride (Novidium, Major and Co.,(Nig.) Ltd., Kano, Nigeria), while the pregnant infected animals were treated after calving.

Trypanosomes Used

The Trypanosoma vivax, Y58 used was isolated from a natural infection in N'dama cow (Leeflang et al., 1976).

Stabilate used was preserved in 5% DMSO and stored in liquid nitrogen. Prior to use in these experiments, the stabilates were first inoculated into sheep which subsequently developed clinical trypanosomiasis. Blood from this sheep was then used to infect the experimental heifers.

Experimental Design

The animals were divided into 3 groups of 6 each and used for the first, second and third trimester trials respectively.

In the first trimester trial, the animals were bred by a proven bull and diagnosed pregnant by day 40. Four of the pregnant animals were inoculated intravenously (via the jugular vein) with approximately $1.5 \times 10^6$ T. vivax strain Y56 on day 60 while the other two pregnant animals were used as controls. The controls were not inoculated. Prior to the infection, all the animals were rectally palpated to ensure that they had normal pregnancy. In addition, these animals were bled and their blood evaluated for baseline hematological data and for the presence of blood parasites.

In the second trimester, 6 four month pregnant animals were used. These animals were also bred as done above. They were diagnosed pregnant on day 40 by rectal examination. In addition, the animals were palpated twice a month until the fourth month when they were used. Four of the animals were inoculated while the other two were used as controls.

In the third trimester, the same procedure was carried out. Four animals were infected while two were used as controls. However, here, two animals were inoculated with T. vivax on the first week of the seventh month while two were inoculated on the 20th day of the seventh month.

Following inoculation, the animals (including the controls) were bled daily for the first 2 weeks and thereafter thrice a week. Serum samples were collected once a week for 3 weeks before the inoculation, on the days of inoculation and once a week thereafter. Blood collections were via the jugular venipuncture into vacutainer tubes with ethylene diaminetetra-acetic acid (EDTA) (1mg/ml blood) as anticoagulant. Samples for sera collection were collected in plain vacutainer tubes without anticoagulant. Such samples were allowed to coagulate at room temperature for 4 hours and then centrifuged at 1000 rpm for 10 minutes at 4°C. The sera were separated out into labelled serum vials and stored at -20°C until they were assayed for the presence of trypanosomes and PCV. On each occasion, rectal temperatures were taken before bleeding.

The clinical changes associated with the infection were noted. In addition, the parturition were also observed; the fetal membranes and the fetus were examined both grossly and histologically.

(a) Hematocrit Centrifuge Test (HCT) for Trypanosomes

A microhematocrit capillary tube containing approximately 70 ul of blood was centrifuge for 5 minutes as for measurement of PCV. The tubes were then placed

on a clean glass slide. A drop of immersion oil was put on top of the capillary tubes with the oil filling the spaces between the capillary tubes. The buffycoat - plasma junction - was then examined under the microscope using the oild immersion objective as described by Woo (1969). The degree of parasitemia was classified as follows:-

\+ = occasional trypanosomes seen

++ = 1 to 2 trypanosomes seen

+++ = 3 to 20 trypanosomes seen

++++ = Over 20 trypanosomes seen

(b) Thin Blood Film Examination for Trypanosome Identification

Clean, grease-free microscope slides were used. About 5 microlitres of blood were placed at one end of the slide and a thin spread made. The slides were quickly air-dried and fixed for 3 minutes in methanol. After this, the slides were stained in 10% Giemsa for 30 minutes, dried and about 100 fields scanned under the oil immersion (x 790).

(c) Packed Cell Volume (PCV)

In order to estimate the degree of anemia, the packed cell volume (PCV) was determined by the micro-hematocrit method using the international microhematocrit centrifuge.

Reproductive and Pregnancy Examination

A reproductive evaluation of all the animals was carried out on the different farms of purchase to ensure that only cycling animals were selected. Also during the 6 months acclimatization period, reproductive examination of all the heifers was done. The heifers were also rectally examined on day 40 after breeding, on the day of infection and twice monthly thereafter. To achieve this, an obstetrical glove was worn and lubricated with soap. The animals were properly restrained in a chute. The hand was then introduced into the rectum and the uterus retracted by the direct method as described by Zemjanis (1970). The ovaries and the genitalia were then palpated. For pregnancy examination, the same procedure was carried out and the uterus and the entire uterine horns palpated as described by Zemjanis (1970).

For the infected animals in the second and third trimesters, the fetuses were palpated to ascertain their viability. The calves were weighed at birth and their blood examined for the presence of trypanosomes. A postmortem examination was carried out on the dead calves and heifer.

Placental Examination

The placentae of the infected animals together with those of the control animals were examined within 4 hours after parturition.

Macroscopic Examination

The placentae were stretched out on a flat board and washed with water. The entire membranes including the cotyledons and the intercotyledonary areas were carefully examined as described by Dennis (1969).

Histological Examination

After the macroscopic examination, sections of the cotyledon, intercotyledonary areas and other parts of the fetal membranes were taken and placed in 10% buffered neutral formalin. They were fixed for 72 hours and then trimmed and processed. Paraffin-embedded sections were cut at 6 microns, stained with hematoxylin and eosin (H & E) and examined microscopically.

Statistical Analysis

Analysis of variance was performed on the PCV and rectal temperatures of the animals in the different groups using the method of Statistical Package for the Social Sciences (SPSS) and the Genstat V method.

Apart from comparing values in the different treatment groups, interaction such as animal/group interactions were considered. A 95 percent confidence level was used thus where $P>0,05$, it was considered non-significant.

# RESULTS

All the inoculated heifers developed clinical trypanosomiasis. There was a gradual deterioration in body condition following the onset of patency. This was characterised by lethargy, weakness, weight loss, dull hair coats and pale mucous membranes. However, this loss of body condition was dependent on the stage ofpregnancy as infected heifers in the third trimester of pregnancy showed more pronounced loss of condition. Infected heifers in the first and second trimester showed a very mild form of the disease. In heifers infected during the third trimester, there was a progressive loss of condition until they delivered either prematurely or full term. All the infected heifers maintained good appetite throughout the duration of the experiment or until death. In all the groups, a chronic course of trypanosomiasis was observed. A summary of the major observations in heifers and calves are shown in Table 8.

Parasitemia:

All the infected heifers developed parasitemia 3-8 days p.i. Initially parasitemia was nigh and fluctuating. Later, it became low and non-detectable about 90 days p.i.

In heifers infected during the first trimester, parasitemia was generally high and fluctuating. In the heifer that aborted, parasitemia was consistently high until 2 days after the abortion. No parasites were found in the blood of the heifer until 25 days abortion when they reappeared.

In the second trimester heifers, parasitemia was sustained though at a comparatively low levels after the first 4 weeks p.i.

Heifers infected during the third trimester had high parasitemia fluctuating between 4+ and 2+ until they delivered or died. Thereafter, parasitemia became low and subsequently, non-detectable (figures 1 - 3).

Rectal Temperatures:

Rectal temperatures increased from the 6th day p.i. in the first and second trimester groups. The increase in the third trimester group was more rapid and increases were recorded from the second day p.i. Highest rectal temperatures recorded in the first, second and third trimester groups were 40.2, 40.3 and 40.3°C respectively.



The increases in rectal temperatures were more pronounced in the infected heifers of the third trimester. This is illustrated in figures 1 - 3. Group variations over the study period were statistically significant ($P<0.05$).

Packed Cell Volume:

In all the groups, there was a progressive decrease in PCV beginning from about the 5th day p.i. The lowest PCV of 11% was recorded in a third trimester heifer on the day of death. The decrease was more pronounced initially in the first trimester heifers as illustrated in tables 1 - 6 and figures 1 - 3. Between the groups, the differences in the PCV as a result of the infection was significant ($P<0.05$). Within the groups, the difference between the PCV of the infected heifers and those of the non-infected controls over the periods was highly significant ($P<0.001$) as show in the analysis of variance table (table 7). In all the heifers (infected and non-infected) there was a decrease in PCV as the animals became full term. After parturition, the animals showed a progressive increase in PCV.

Effects on Pregnancy and Delivery:

In the first trimester, one heifer (No. 1933) aborted 39 days post infection and 99 days post breeding. The aborted fetus was not seen; only parts of the fetal membranes were seen hanging down the vulva. The remaining three infected heifers together with the two controls had relatively normal pregnancy and delivery. In heifer number 1703, extensive vulva edema was observed 56 days p.i. (figure 6). The swelling was massive and by the end of the second day, it started to disappear. This same heifer had an extensive ventral edema 2 weeks before parturition. The navel area was mostly involved and pre-parturient udder enlargement was significantly larger than those of other infected heifers. No dystocia case was observed and fetal membranes were dropped within 4 hours postpartum.

In the second trimester, the T. vivax infection did not seem to have affected the pregnancy. All the infected heifers had normal gestation and calved without problems. Here again, there was no retained placenta problem. The placentae in all dropped within 4 hours postpartum.

In the pregnant heifers infected during the third trimester, the effect of the infection on pregnancy was dramatic as the heifers started "throwing out" the fetuses prematurely. Premature delivery occurred in 3 (75%) out of the 4 infected heifers. In the 2 heifers infected on day 10 of the seventh month, they were overwhelmed by the infection and premature delivery occurred 13 days and 23 days respectively p.i. In the first case, the calf died within 20 minutes after delivery (figure 8) the second calf survived even though it had parasitemia of 3+ (figure 9). One of the two remaining infected heifers had a premature delivery while the other could not go into labor at term and the calf had to be pulled out (figure 7). The heifer had been recumbent 5 days before the calf was pulled out and it died about 3 hours after the forced extraction. The heifer died of the experimental infection with T. vivax as shown by the postmortem examination. The calf lived for less than 24 hours. In the 3 heifers that survived, 2 had retained placentae which were mannually removed after 48 hours. Uterine involution was delayed in both.

Effect on the Calves:

The birth weights of the calves for the first, second and third trimester infected heifers ranged from 18 - 24; 20 - 28 and 10 - 18 kg respectively. The calves of heifers infected during the first trimester were weaker than those of the second trimester or the controls. Although they were full term, they required good nursing to survive.

In all the calves that survived, none showed clinical trypanosomiasis. In the calves of heifers infected during the third trimester, only one showed T. vivax in its blood and had a high parasitemia of 3+. No clinical signs of the infection were developed by the calf apart from swollen lymph nodes. There was a progressive decrease in the parastemia until parasites were no longer demonstrable in the blood about 64 days post delivery.

The PCV of calves of the infected heifers at birth are as shown:

First trimester: 27.5 - 36.5%

Second trimester: 30 - 37%

Third trimester: 22.5 - 31.0%

For the first 6 days postpartum there were progressive decreases in the PCVs. Thereafter the PCVs increased steadily until about 30 days later when a rather steady PCV of about 35% was maintained.

Gross and Histological Findings:

The placentae in all the infected heifers did not show any gross abnormalities - (figure 12). Histological sections did not reveal much either. However in the placentae of heifers infected during the third trimester, it was observed that there were more mononuclear cells in the sections than in those of the controls (figure 4 & 5).

In the two calves that died, postmortem results revealed that the lymph nodes were enlarged. In both cases, the spleens were grossly enlarged also. There were petechial hemorrhages in the hearts. In the calf that died soon after birth (figure 8), 35mls of blood tinged fluid were obtained from the peritoneal cavity. There was also some hemothorax. In the other calf, 15 mls of blood tinged fluid were removed from the pericadial sac and 210mls of same fluid from the thoracic cavity. In both calves, there was serus (gelatinous) atrophy of perirenal and pelvic fats.

Histological sections of organs from the calves revealed inconclusive evidence of some hemorrhage, cardiac muscle degeneration and edema. It must be emphasized however, that the heart muscles of the two calves had the same lesions (figure 10 & 11).

# DISCUSSION

Cattle generally show the characteristic signs of trypanosomiasis when infected experimentally with T. vivax. In pregnant heifers, the signs, especially emaciation and lethargy are more pronounced during the third trimester as shown in this experiment. This might be due to increased stress at that period of pregnancy.

Similarly, the observed high parasitemia in the third trimester heifers are also due to the same reason. Thus when parturition occurs either prematurely or full term, the stress of pregnancy is removed and the animals were therefore better able to fight the infection. The results of the PCV in the infected heifers are more difficult to interprete. It is not known why heifers in the first trimester had more severe anemia than those of the second and even third trimaster infected heifers. It may be associated with the endocrine changes associated with pregnancy.

The abortion observed in heifer No. 1933 infected during the first trimester may be due to the infection with T. vivax. Why only one out of the 4 infected heifers should abort could be due to individual variation in response to trypanosome infection.

Murray et al., (1981) found differences in susceptibility to trypanosome infection not only between breeds but also within breeds. There was also individual variations in temperature, PCV and parasitemia (figures 1, 2 & 3). Bovine abortion due to trypanosomiasis during the first trimester is a common observation among the Fulani cattle owners in Nigeria. Similar observation has been made by Murray et al., (1981).

That none of the heifers infected during the second trimester aborted was rather interesting. This is in accordance with the mild clinical manifestations of the infection shown by this group of heifers.

Although abortions, premature delivery, still birth and weak calf deliveries as seen in the infected third trimester heifers have for long been associated with bovine trypanosomiasis, the experiments reported here have provided some explanation for these. This explanation is that during the migration to the southern guinea zones, the tsetsefly areas, some of the cows already heavily pregnant get infected and subsequently have still births or deliver prematurely, or deliver weak calves. Some of the cows that are then in the second trimester stage of pregnancy resist the infection and carry their pregnancy to term and give birth to normal calves, usually on their return to their

normal breeding zones. However some cows and heifers that get infected during their first trimester stage of pregnancy in the tse-tse fly belt abort subsequently.

The occurrence of abortion in early and late pregnancy has also been observed by Murray et al. (1981) in Zebu cattle. However this is the first experimental T. vivax infection resulting in premature delivery in pregnant heifers. This concludes that transplacental transmission of T. vivax does occur.

Although several workers have reported abortion in cows associated with trypanosome infection (Hornby 1921; Pigneur, 1932; Dikmans et al.. 1957; Woo and Limebeer, 1971; Murray et al., 1981) nobody has been able to show conclusively the mechanism (s) involved in this abortion. The results of these present experiments show that there are no gross but minor and inconclusive histological placental lesions. The observed increase in mononuclear cells may be an indication of inapparent inflammatory reaction caused by T. vivax migration through the organ. More work is definitely required in this area.

While the observed fetal heart muscle degeneration and edema have also been reported in calves experimentally infected with T. congolense (Valli and Forsbery, 1979) no such finding had earlier been associated with

T. vivax infection in calves. It must be emphasized here that no T. vivax was found in these particular calves reported in these experiments at the time of birth. Conversely in one of the premature calves that survived T. vivax was recovered from it throughout a period of over 64 days without clinical manifestations.

This might be due to the mind strain of T. vivax used in this experiment (Ilemobade, personal communication) and the fact that calves in general are known to be resistant to trypanosome infection (Fiennes, 1970).

The observation that pregnant heifers infected during the second trimester did not abort may indicate that, somehow, heifers at this stage of pregnancy have better resistance to trypanosomiasis, thereby preventing transplacental transmission and subsequently fetal infection. Alternatively, it may mean that fetuses at this stage of development are able to resist the infection having acquired immunocompetence to fight the infection. Similarly, fetuses infected during the third trimester have some of their organs e.g. heart muscle damaged. Some or most of the fetuses survive but are stressed. The stress results in the production of corticosteroid from the rather matured

fetal adrenal glands. These hormones then act on the fetoplacental unit interrupting its production of luteotrophic hormones and progresterone as shown by Osburn and Kennedy (1981). This is then complicated by the rather severe maternal infection, hyperthermia and fetal hypoxia resulting from maternal anemia caused by the infection. The result is the premature expulsion of the fetus. The occasional early abortion observed may be due to fetal infection. Since susceptibility varies between individuals, some of the infected fetuses die and are expelled by the dam while most survive and gradually get rid of the infection before birth. The observed weakness of calves in this category during the experiment may not be unconnected with the early damage to the spleen.

In conclusion, it may be stated that in pregnant heifers infected with T. vivax, transplacental transmission of the organism occurs, resulting in fetal infection. The outcome of the fetal infection then depends mainly on the stage of the pregnancy and to a lesser extent on the variation in individual susceptibility.

## SUMMARY

A group of 18 heifers purchased from the Kano State Livestock Investigation and Breeding Center (LIBC) in Gumel and from the National Animal Production Research Institute (NAPRI) Shika, Kaduna State was tested for brucellosis, dewormed and the baseline hematological data determined. The heifers were fed grass hay supplemented with cotton seed cake and wheat offals. Following 6 months acclimatization in the Faculty of Veterinary Medicine, Ahmadu Bello University, Zaria research pens, they were then bred by a proven bull and divided into 3 groups of 6 heifers each.

In the first group, 4 heifers were infected with Trypanosoma vivax on day 60 (first trimester) of pregnancy while 2 other pregnant heifers were uninfected controls. The second and third groups were similarly infected in the 4th (second trimester) and 7th (third trimester) months of pregnancy.

One infected heifer in the first trimester group aborted 39 days post infection (p.i.) while the remaining 3 had relatively normal gestation and parturition. In the second trimester group, the pregnancies were carried to term with normal deliveries.

In the third trimester group, 3 infected heifers (75%) had premature deliveries while the 4th died about 3 hours after the full term calf was pulled out. All the control heifers had normal gestation and parturition.

No gross abnormalities were seen in the placentae of the infected heifers but histological sections of the infected third trimester pregnancy heifers revealed more mononuclear cells than in those of the uninfected controls. Post-mortem examination of the dead premature calves showed lymphoadenopathy, spleenomegally, serous atrophy of perirenal and pelvic fat, epicardial petechial hemorrhage and blood-tinged peritoneal and pericardial fluids. Histologically, there were slight myocardial hemorrhages and edema.

T. vivax was recovered in the blood of one of the premature calves. Both birth weights and PCV of the calves were affected by the experimental maternal infection. The birth-weights and PCV of calves of infected dams had lower birth-weights and PCV than the calves of the control heifers. This work therefore demonstrates transplacental transmission of T. vivax in heifer.

Amongst the infected heifers in the 3 stages of pregnancy, clinical manifestation of the infection varied with heifers in the third trimester showing

more severe signs than those in the first and second trimesters of pregnancy. There is also group variations in rectal temperature and PCV of the infected heifers.

Table 1: Means and standard deviations of PCV and rectal temperatures of heifers infected during the first trimester

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| -8 | 34.0 ± 6.27 | 38.7 ± 0.24 |
| -3 | 34.0 ± 3.75 | 38.7 ± 0.42 |
| 0 | 33.9 ± 3.25 | 38.5 ± 0.66 |
| 1 | 34.0 ± 3.50 | 38.5 ± 0.19 |
| 2 | 33.5 ± 3.70 | 38.8 ± 0.39 |
| 3 | 32.75 ± 3.75 | 38.8 ± 0.32 |
| 4 | 33.25 ± 4.51 | 38.6 ± 0.15 |
| 5 | 33.25 ± 4.51 | 38.8 ± 0.23 |
| 6 | 30.87 ± 3.75 | 38.8 ± 0.49 |
| 7 | 30.25 ± 4.51 | 38.4 ± 0.75 |
| 8 | 28.75 ± 4.33 | 38.9 ± 0.53 |
| 9 | 28.50 ± 3.69 | 38.9 ± 0.80 |
| 10 | 27.87 ± 3.96 | 39.1 ± 0.52 |
| 11 | 27.37 ± 4.78 | 39.2 ± 0.49 |
| 12 | 28.37 ± 5.34 | 39.1 ± 0.65 |
| 13 | 28.37 ± 5.85 | 39.4 ± 0.61 |
| 14 | 27.37 ± 3.68 | 39.2 ± 0.43 |
| 15 | 25.83 ± 3.01 | 39.6 ± 0.52 |
| 16 | 25.83 ± 3.01 | 39.0 ± 0.35 |
| 17 | 24.72 ± 5.34 | 38.8 ± 0.29 |
| 18 | 26.75 ± 2.59 | 38.4 ± 0.53 |
| 19 | 26.75 ± 2.22 | 38.8 ± 0.86 |
| 21 | 26.25 ± 4.66 | 39.2 ± 0.40 |
| 25 | 26.13 ± 6.11 | 38.8 ± 0.39 |
| 28 | 25.62 ± 2.62 | 38.8 ± 0.68 |
| 31 | 26.87 ± 4.17 | 39.6 ± 0.63 |
| 36 | 26.50 ± 4.17 | 39.2 ± 0.17 |
| 39 | 26.50 ± 4.43 | 38.8 ± 0.29 |
| 42 | 25.37 ± 4.67 | 39.2 ± 0.34 |
| 44 | 28.37 ± 6.47 | 38.6 ± 0.19 |
| 49 | 28.25 ± 5.04 | 39.1 ± 0.43 |
| 51 | 27.62 ± 2.56 | 38.8 ± 0.42 |
| 53 | 28.62 ± 2.86 | 38.7 ± 0.28 |
| 58 | 29.12 ± 3.11 | 38.8 ± 0.48 |
| 60 | 30.50 ± 2.48 | 38.8 ± 0.26 |
| 63 | 28.75 ± 3.01 | 38.5 ± 0.06 |
| 65 | 29.75 ± 2.36 | 38.4 ± 0.05 |
| 70 | 29.00 ± 3.85 | 38.6 ± 0.2 |
| 77 | 29.37 ± 5.18 | 38.5 ± 0.30 |
| 79 | 31.00 ± 2.34 | 38.7 ± 0.45 |
| 81 | 30.00 ± 2.00 | 38.4 ± 0.95 |
| 86 | 30.25 ± 2.63 | 38.5 ± 0.10 |
| 88 | 30.62 ± 2.25 | 38.5 ± 0.10 |
| 91 | 30.37 ± 2.56 | 38.5 ± 0.05 |
| 93 | 30.37 ± 2.49 | 38.5 ± 0.13 |
| 95 | 30.50 ± 1.91 | 38.6 ± 0.15 |

| Time (days) | PCV % | Temperature ($^{O}$C) |
|---|---|---|
| 98 | 30.75 ± 2.32 | 38.6 ± 0.22 |
| 100 | 31.62 ± 3.54 | 38.5 ± 0.05 |
| 102 | 30.50 ± 4.02 | 38.5 ± 0.17 |
| 109 | 30.12 ± 2.65 | 38.5 ± 0.05 |
| 115 | 29.75 ± 2.72 | 38.5 ± 0.08 |
| 122 | 30.25 ± 2.500 | 38.5 ± 0.13 |
| 129 | 30.25 ± 2.72 | 38.5 ± 0.12 |

Table 2: Means and standard deviations of PCV and rectal temperatures of heifers infected during the second trimester

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| -8 | 38.25 ± 3.28 | 38.65 ± 0.12 |
| -3 | 37.25 ± 2.66 | 38.37 ± 0.32 |
| 0 | 37.50 ± 2.38 | 38.52 ± 0.09 |
| 1 | 37.37 ± 2.28 | 38.45 ± 0.05 |
| 2 | 37.37 ± 2.28 | 38.42 ± 0.05 |
| 3 | 36.87 ± 2.66 | 38.47 ± 0.05 |
| 4 | 36.38 ± 3.68 | 38.87 ± 0.57 |
| 5 | 34.87 ± 3.75 | 38.90 ± 0.42 |
| 6 | 34.87 ± 5.12 | 38.90 ± 0.49 |
| 7 | 33.50 ± 4.26 | 39.20 ± 0.48 |
| 8 | 33.00 ± 4.79 | 39.17 ± 0.54 |
| 9 | 32.25 ± 3.96 | 39.10 ± 0.28 |
| 10 | 32.13 ± 4.17 | 39.47 ± 0.09 |
| 11 | 30.95 ± 3.85 | 39.57 ± 0.43 |
| 12 | 29.00 ± 2.71 | 39.45 ± 0.33 |
| 13 | 29.63 ± 3.09 | 39.42 ± 0.36 |
| 14 | 29.75 ± 2.06 | 39.45 ± 0.49 |
| 15 | 30.63 ± 3.57 | 39.45 ± 0.35 |
| 16 | 29.50 ± 2.74 | 39.30 ± 1.00 |
| 17 | 29.63 ± 1.65 | 39.17 ± 0.51 |
| 18 | 28.00 ± 1.96 | 39.37 ± 0.35 |
| 19 | 28.25 ± 3.30 | 39.52 ± 0.37 |
| 21 | 28.00 ± 2.94 | 38.85 ± 0.70 |
| 25 | 27.37 ± 2.28 | 38.85 ± 0.50 |
| 28 | 27.37 ± 2.28 | 38.85 ± 0.50 |
| 31 | 26.37 ± 4.30 | 39.35 ± 0.76 |
| 36 | 26.87 ± 4.94 | 39.23 ± 0.20 |
| 39 | 25.25 ± 4.86 | 39.00 ± 0.01 |
| 42 | 23.63 ± 5.38 | 39.23 ± 0.34 |
| 44 | 25.75 ± 4.99 | 39.05 ± 0.33 |
| 49 | 27.00 ± 4.76 | 38.95 ± 0.65 |
| 51 | 27.25 ± 2.47 | 39.10 ± 0.25 |
| 53 | 27.37 ± 2.49 | 39.28 ± 0.26 |
| 58 | 27.75 ± 3.96 | 38.70 ± 0.47 |
| 60 | 26.00 ± 6.69 | 38.80 ± 0.54 |
| 63 | 25.88 ± 5.97 | 38.65 ± 0.73 |
| 65 | 27.12 ± 4.44 | 38.85 ± 0.64 |
| 70 | 26.12 ± 4.21 | 38.55 ± 0.17 |
| 77 | 25.00 ± 4.14 | 38.60 ± 0.14 |
| 79 | 24.13 ± 3.92 | 38.57 ± 0.22 |
| 81 | 24.25 ± 4.50 | 38.50 ± 0.08 |
| 86 | 18.28 ± 11.65 | 38.55 ± 0.17 |
| 88 | 26.38 ± 2.78 | 38.90 ± 0.08 |
| 91 | 26.63 ± 1.11 | 38.83 ± 0.71 |
| 93 | 26.12 ± 1.65 | 38.65 ± 0.13 |
| 95 | 26.00 ± 2.12 | 38.52 ± 0.09 |

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| 98 | 27.13 ± 1.65 | 38.53 ± 0.09 |
| 100 | 27.75 ± 1.71 | 38.50 ± 0.14 |
| 102 | 28.75 ± 1.66 | 39.43 ± 0.09 |
| 109 | 29.37 ± 0.48 | 38.48 ± 0.05 |
| 115 | 29.50 ± 1.00 | 38.43 ± 0.09 |
| 122 | 30.00 ± 0.91 | 38.40 ± 0.36 |
| 129 | 30.00 ± 0.81 | 38.50 ± 0.34 |

Table 3: Means and standard deviations of PCV and rectal temperatures of heifers infected during the third trimester

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| -8 | 36.87 ± 4.15 | 38.35 ± 0.35 |
| -3 | 36.75 ± 4.63 | 38.60 ± 0.28 |
| 0 | 36.63 ± 5.13 | 38.50 ± 0.13 |
| 1 | 36.62 ± 4.69 | 38.70 ± 0.22 |
| 2 | 35.00 ± 4.30 | 39.80 ± 0.37 |
| 3 | 36.75 ± 5.08 | 39.80 ± 0.34 |
| 4 | 37.13 ± 4.30 | 39.65 ± 0.37 |
| 5 | 34.00 ± 2.45 | 39.55 ± 0.54 |
| 6 | 33.50 ± 3.42 | 39.53 ± 0.45 |
| 7 | 32.37 ± 1.60 | 39.77 ± 0.26 |
| 8 | 29.50 ± 3.34 | 39.50 ± 0.41 |
| 9 | 28.63 ± 3.90 | 39.53 ± 0.26 |
| 10 | 28.75 ± 4.11 | 39.70 ± 0.22 |
| 11 | 28.38 ± 4.42 | 39.55 ± 0.40 |
| 12 | 27.63 ± 4.11 | 39.45 ± 0.33 |
| 13 | 26.88 ± 3.56 | 39.80 ± 0.20 |
| 14 | 26.13 ± 3.28 | 39.70 ± 0.18 |
| 15 | 25.00 ± 3.98 | 39.65 ± 0.17 |
| 16 | 25.13 ± 3.57 | 39.20 ± 0.28 |
| 17 | 25.50 ± 0.71 | 39.00 ± 0.24 |
| 18 | 23.37 ± 5.02 | 38.83 ± 0.49 |
| 19 | 24.13 ± 7.44 | 38.70 ± 0.36 |
| 21 | 23.33 ± 7.28 | 38.70 ± 0.36 |
| 25 | 24.38 ± 7.23 | 38.35 ± 0.24 |
| 28 | 25.50 ± 5.12 | 38.85 ± 0.47 |
| 31 | 25.62 ± 5.72 | 38.87 ± 0.49 |
| 36 | 26.13 ± 6.59 | 38.85 ± 0.35 |
| 39 | 27.00 ± 4.65 | 38.83 ± 0.53 |
| 42 | 27.50 ± 3.93 | 38.85 ± 0.54 |
| 44 | 28.00 ± 6.41 | 38.93 ± 0.57 |
| 49 | 25.13 ± 5.89 | 38.57 ± 0.45 |
| 51 | 25.75 ± 8.74 | 38.20 ± 0.82 |
| 53 | 24.75 ± 7.39 | 38.56 ± 0.12 |
| 58 | 24.87 ± 8.23 | 38.67 ± 0.29 |
| 60 | 23.75 ± 7.41 | 39.10 ± 0.46 |
| 63 | 24.00 ± 8.51 | 39.54 ± 0.36 |
| 65 | 22.00 ± 9.14 | 38.90 ± 0.36 |
| 70 | 19.88 ± 6.76 | 38.87 ± 0.23 |
| 77 | 18.75 ± 7.14 | 38.66 ± 0.23 |
| 79 | 23.00 ± 6.95 | 38.50 ± 0.10 |
| 81 | 22.83 ± 7.69 | 38.50 ± 0.10 |
| 86 | 25.83 ± 6.01 | 38.63 ± 0.10 |
| 88 | 28.17 ± 3.25 | 38.90 ± 0.53 |
| 91 | 26.83 ± 3.25 | 38.90 ± 0.53 |

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| 93 | 28.16 ± 2.75 | 38.63 ± 0.23 |
| 95 | 28.50 ± 2.29 | 38.50 ± 0.45 |
| 100 | 28.83 ± 3.82 | 38.50 ± 0.45 |
| 102 | 28.16 ± 2.75 | 38.50 ± 0.82 |
| 109 | 30.50 ± 2.29 | 38.50 ± 0.35 |
| 115 | 28.16 ± 2.25 | 38.50 ± 0.012 |
| 122 | 26.16 ± 2.25 | 38.50 ± 0.23 |
| 129 | 28.50 ± 0.50 | 38.45 ± 0.37 |

Table 4: Means and standard deviations of PCV and rectal temperatures of control heifers for first trimester trial

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| -8 | 39.75 ± 10.25 | 38.5 ± 0.00 |
| -3 | 39.50 ± 9.89 | 38.5 ± 0.07 |
| 0 | 39.25 ± 10.25 | 38.50 ± 0.00 |
| 1 | 39.00 ± 9.89 | 38.50 ± 0.00 |
| 2 | 40.50 ± 10.61 | 38.50 ± 0.00 |
| 3 | 39.50 ± 9.19 | 38.55 ± 0.07 |
| 4 | 39.50 ± 8.48 | 38.55 ± 0.07 |
| 5 | 38.50 ± 8.49 | 38.60 ± 0.00 |
| 6 | 38.50 ± 8.49 | 38.55 ± 0.07 |
| 7 | 38.25 ± 8.13 | 38.45 ± 0.07 |
| 8 | 38.50 ± 7.78 | 38.55 ± 0.07 |
| 9 | 40.00 ± 9.89 | 38.50 ± 0.00 |
| 10 | 39.75 ± 8.83 | 38.50 ± 0.00 |
| 11 | 39.50 ± 9.19 | 38.50 ± 0.00 |
| 12 | 38.75 ± 8.83 | 38.55 ± 0.07 |
| 13 | 38.50 ± 9.19 | 38.55 ± 0.07 |
| 14 | 39.25 ± 8.83 | 38.55 ± 0.07 |
| 15 | 39.50 ± 7.77 | 38.50 ± 0.00 |
| 16 | 39.00 ± 6.36 | 38.50 ± 0.00 |
| 17 | 38.25 ± 6.71 | 38.55 ± 0.07 |
| 18 | 39.00 ± 7.07 | 38.50 ± 0.00 |
| 19 | 39.00 ± 6.71 | 38.55 ± 0.07 |
| 21 | 38.25 ± 6.71 | 38.50 ± 0.00 |
| 25 | 38.50 ± 7.71 | 38.50 ± 0.00 |
| 25 | 37.25 ± 6.71 | 38.45 ± 0.07 |
| 31 | 37.50 ± 7.77 | 38.50 ± 0.00 |
| 36 | 38.00 ± 7.07 | 38.55 ± 0.07 |
| 39 | 39.75 ± 4.59 | 38.55 ± 0.07 |
| 42 | 36.75 ± 8.84 | 38.55 ± 0.07 |
| 44 | 38.00 ± 9.89 | 38.50 ± 0.00 |
| 49 | 36.25 ± 5.30 | 38.55 ± 0.07 |
| 51 | 37.25 ± 7.42 | 38.50 ± 0.00 |
| 53 | 37.25 ± 7.42 | 38.45 ± 0.07 |
| 58 | 37.25 ± 7.42 | 38.55 ± 0.07 |
| 60 | 37.25 ± 7.42 | 38.50 ± 0.00 |
| 63 | 37.00 ± 7.07 | 38.55 ± 0.07 |
| 65 | 36.77 ± 8.13 | 38.50 ± 0.00 |
| 70 | 37.00 ± 7.07 | 38.55 ± 0.07 |
| 77 | 37.00 ± 8.48 | 38.50 ± 0.00 |
| 79 | 38.50 ± 6.36 | 38.50 ± 0.00 |
| 81 | 39.25 ± 8.13 | 38.45 ± 0.07 |
| 86 | 38.50 ± 7.77 | 38.60 ± 0.00 |
| 88 | 38.50 ± 7.77 | 38.50 ± 0.00 |
| 91 | 38.50 ± 7.77 | 38.50 ± 0.00 |

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| 93 | 37.25 ± 6.71 | 38.50 ± 0.00 |
| 95 | 37.50 ± 3.53 | 38.55 ± 0.07 |
| 100 | 37.50 ± 6.36 | 38.55 ± 0.07 |
| 102 | 38.50 ± 4.94 | 38.50 ± 0.00 |
| 109 | 37.75 ± 7.42 | 38.40 ± 0.28 |
| 115 | 38.00 ± 7.07 | 38.60 ± 0.14 |
| 122 | 38.75 ± 7.42 | 38.50 ± 0.00 |
| 129 | 38.50 ± 7.77 | 38.55 ± 0.07 |

Table 5: Means and standard deviations of PCV and rectal temperatures of control heifers for the second trimester trial

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| -8 | 35.75 ± 4.59 | 38.55 ± 0.07 |
| -3 | 37.00 ± 5.65 | 38.40 ± 0.00 |
| 0 | 35.75 ± 4.59 | 38.55 ± 0.21 |
| 1 | 37.75 ± 7.42 | 38.40 ± 0.14 |
| 2 | 37.50 ± 7.07 | 38.60 ± 0.00 |
| 3 | 37.50 ± 7.07 | 38.40 ± 0.14 |
| 4 | 36.50 ± 4.94 | 38.50 ± 0.00 |
| 5 | 36.00 ± 5.65 | 38.60 ± 0.14 |
| 6 | 36.00 ± 5.65 | 38.45 ± 0.07 |
| 7 | 36.00 ± 5.65 | 38.55 ± 0.07 |
| 8 | 37.50 ± 6.36 | 38.45 ± 0.07 |
| 9 | 37.00 ± 5.65 | 38.60 ± 0.14 |
| 10 | 36.25 ± 5.30 | 38.55 ± 0.07 |
| 11 | 36.25 ± 5.30 | 38.40 ± 0.14 |
| 12 | 36.00 ± 5.65 | 38.45 ± 0.07 |
| 13 | 35.25 ± 5.30 | 38.55 ± 0.07 |
| 14 | 35.25 ± 5.65 | 38.45 ± 0.07 |
| 15 | 35.50 ± 5.65 | 38.50 ± 0.00 |
| 16 | 34.50 ± 4.94 | 38.50 ± 0.14 |
| 17 | 35.00 ± 7.07 | 38.50 ± 0.00 |
| 18 | 36.00 ± 4.24 | 38.50 ± 0.00 |
| 19 | 35.25 ± 3.88 | 38.50 ± 0.00 |
| 21 | 35.00 ± 6.36 | 38.50 ± 0.00 |
| 25 | 35.00 ± 6.36 | 38.50 ± 0.00 |
| 28 | 35.50 ± 5.65 | 38.50 ± 0.00 |
| 31 | 35.75 ± 5.30 | 38.70 ± 0.00 |
| 36 | 35.75 ± 5.30 | 38.55 ± 0.07 |
| 39 | 35.75 ± 5.30 | 38.35 ± 0.07 |
| 42 | 35.75 ± 5.30 | 38.75 ± 0.05 |
| 44 | 35.75 ± 5.30 | 38.55 ± 0.07 |
| 49 | 36.75 ± 5.30 | 38.40 ± 0.14 |
| 51 | 37.00 ± 5.65 | 38.25 ± 0.35 |
| 53 | 36.25 ± 5.30 | 38.55 ± 0.07 |
| 56 | 36.00 ± 4.94 | 38.65 ± 0.07 |
| 60 | 36.25 ± 5.30 | 38.55 ± 0.07 |
| 63 | 35.25 ± 4.59 | 38.50 ± 0.00 |
| 65 | 35.50 ± 4.94 | 38.50 ± 0.00 |
| 70 | 35.50 ± 4.94 | 38.50 ± 0.00 |
| 77 | 35.75 ± 5.30 | 38.50 ± 0.00 |
| 79 | 36.00 ± 5.65 | 38.40 ± 0.14 |
| 81 | 37.50 ± 7.77 | 38.50 ± 0.00 |
| 86 | 37.75 ± 6.01 | 38.45 ± 0.07 |
| 88 | 37.00 ± 6.77 | 38.50 ± 0.00 |
| 91 | 36.25 ± 5.83 | 38.50 ± 0.00 |
| 93 | 35.75 ± 4.39 | 38.50 ± 0.00 |

| Time (days) | ECV % | Temperature (°C) |
|---|---|---|
| 95 | 35.25 ± 4.59 | 32.45 ± 0.07 |
| 70) | 34.50 ± 6.36 | 32.55 ± 0.07 |
| 102 | 34.75 ± 6.71 | 30.50 ± 0.00 |
| 103 | 34.75 ± 6.71 | 32.50 ± 0.00 |
| 75 | 35.00 ± 7.07 | 35.50 ± 0.00 |
| 120 | 36.00 ± 7.07 | 34.45 ± 0.07 |
| 129 | 36.50 ± 7.77 | 32.50 ± 0.00 |

Table 6: Means and standard deviations of PCV and rectal temperatures of control heifers for third trimester trial

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| -8 | 31.25 ± 0.35 | 38.30 ± 0.0 |
| -3 | 31.75 ± 1.76 | 38.60 ± 0.14 |
| 0 | 32.75 ± 1.76 | 38.55 ± 0.07 |
| 1 | 32.00 ± 1.41 | 38.60 ± 0.14 |
| 2 | 31.25 ± 2.42 | 38.55 ± 0.21 |
| 3 | 31.25 ± 2.47 | 38.40 ± 0.14 |
| 4 | 31.50 ± 2.12 | 38.25 ± 0.35 |
| 5 | 31.75 ± 2.47 | 38.35 ± 0.35 |
| 6 | 31.25 ± 2.47 | 38.35 ± 0.07 |
| 7 | 32.25 ± 3.88 | 38.30 ± 0.00 |
| 8 | 34.50 ± 0.75 | 38.30 ± 0.00 |
| 9 | 32.00 ± 3.54 | 38.55 ± 0.07 |
| 10 | 31.75 ± 3.88 | 38.45 ± 0.07 |
| 11 | 31.00 ± 3.04 | 38.33 ± 0.11 |
| 12 | 30.75 ± 5.30 | 38.45 ± 0.07 |
| 13 | 30.00 ± 2.82 | 38.50 ± 0.21 |
| 14 | 30.50 ± 3.54 | 38.50 ± 0.00 |
| 15 | 31.00 ± 0.71 | 38.55 ± 0.07 |
| 16 | 30.00 ± 0.72 | 38.55 ± 0.07 |
| 17 | 30.50 ± 1.41 | 38.55 ± 0.07 |
| 18 | 31.00 ± 1.76 | 38.55 ± 0.07 |
| 19 | 29.50 ± 2.82 | 38.45 ± 0.21 |
| 21 | 29.00 ± 2.12 | 38.25 ± 0.07 |
| 25 | 29.50 ± 3.53 | 38.35 ± 0.07 |
| 28 | 28.50 ± 2.12 | 38.40 ± 0.00 |
| 31 | 30.00 ± 2.83 | 38.35 ± 0.21 |
| 36 | 29.00 ± 2.83 | 38.55 ± 0.07 |
| 39 | 30.00 ± 2.83 | 38.40 ± 0.14 |
| 42 | 30.50 ± 2.83 | 38.45 ± 0.07 |
| 44 | 30.50 ± 3.18 | 38.40 ± 0.00 |
| 49 | 30.75 ± 3.18 | 38.55 ± 0.07 |
| 51 | 30.00 ± 4.24 | 38.45 ± 0.07 |
| 53 | 30.50 ± 3.53 | 38.40 ± 0.14 |
| 58 | 29.25 ± 1.77 | 38.40 ± 0.14 |
| 60 | 29.00 ± 1.41 | 38.35 ± 0.07 |
| 63 | 29.75 ± 1.77 | 38.40 ± 0.14 |
| 65 | 29.25 ± 0.35 | 38.30 ± 0.14 |
| 70 | 29.50 ± 0.71 | 38.40 ± 0.14 |
| 77 | 29.00 ± 0.00 | 38.40 ± 0.00 |
| 79 | 28.50 ± 1.41 | 38.40 ± 0.00 |
| 81 | 28.25 ± 2.47 | 38.40 ± 0.14 |
| 88 | 28.50 ± 2.12 | 38.50 ± 0.28 |
| 91 | 29.25 ± 1.77 | 38.40 ± 0.00 |
| 93 | 29.00 ± 1.41 | 38.40 ± 0.28 |

| Time (days) | PCV % | Temperature (°C) |
|---|---|---|
| 95 | 29.25 ± 2.47 | 30.60 ± 0.14 |
| 100 | 29.25 ± 2.47 | 30.55 ± 0.00 |
| 102 | 30.50 ± 3.55 | 30.55 ± 0.21 |
| 109 | 30.50 ± 3.53 | 30.50 ± 0.00 |
| 115 | 30.50 ± 3.53 | 30.50 ± 0.00 |
| 122 | 33.66 ± 6.02 | 30.45 ± 0.07 |
| 129 | 33.66 ± 6.02 | 30.55 ± 0.07 |

Table 7: Analysis of variance table for packed cell volume (PCV)

| Source of variation | DF | SS | SS% | MS | VR |
|---|---|---|---|---|---|
| Group | 3 | 4802.68 | 7.45 | 1600.89 | 95.416 |
| Period | 58 | 12833.28 | 19.92 | 225.15 | 13.42 |
| Animal | 5 | 18577.10 | 28.83 | 3715.42 | 221.445 |
| Group/Animal Interaction | 15 | 9242.82 | 14.34 | 616.19 | 36.726 |
| Residual | 1284 | 21543.07 | 33.43 | 16.78 | |

Table 6: Summary of clinical observations in infected heifers and clinical/pathological observations in the calves

| | Groups of infected pregnant heifers | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| Highest rectal temperature in infected heifers (°C) | 40.2 | 40.3 | 40.3 |
| Lowest PCV recorded in infected heifers (%) | 20.0 | 16.0 | 11.0 |
| Nature of infection in heifers | mild | mild | severe |
| Abortion | + | - | - |
| Premature delivery | - | - | + |
| Condition of calves at birth | weak | viable | weak |
| Average weights of calves at birth (kg) | 20.7 | 28.4 | 15.0 |
| Average PCV of calves at birth (%) | 30.3 | 31.5 | 25.6 |
| Death of calves soon after birth | - | - | + |
| Retained Placenta | - | - | + |
| Increased monnuclear cells in histological section of the placenta | -- | - | + |
| Cardiac lesions in the fetus | NA | NA | + |

\+ = Present
\- = Absent
NA = Not Applicable

1 = heifers infected during the first trimester.. N = 4
2 = heifers infected during the second trimester. N = 4
3 = heifers infected during the third trimester. N = 4

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Figure 1

Temperature curve, parasitemia and packed cell volume (PCV) in Zebu heifers infected with Trypanosoma vivax during the first trimester of pregnancy.

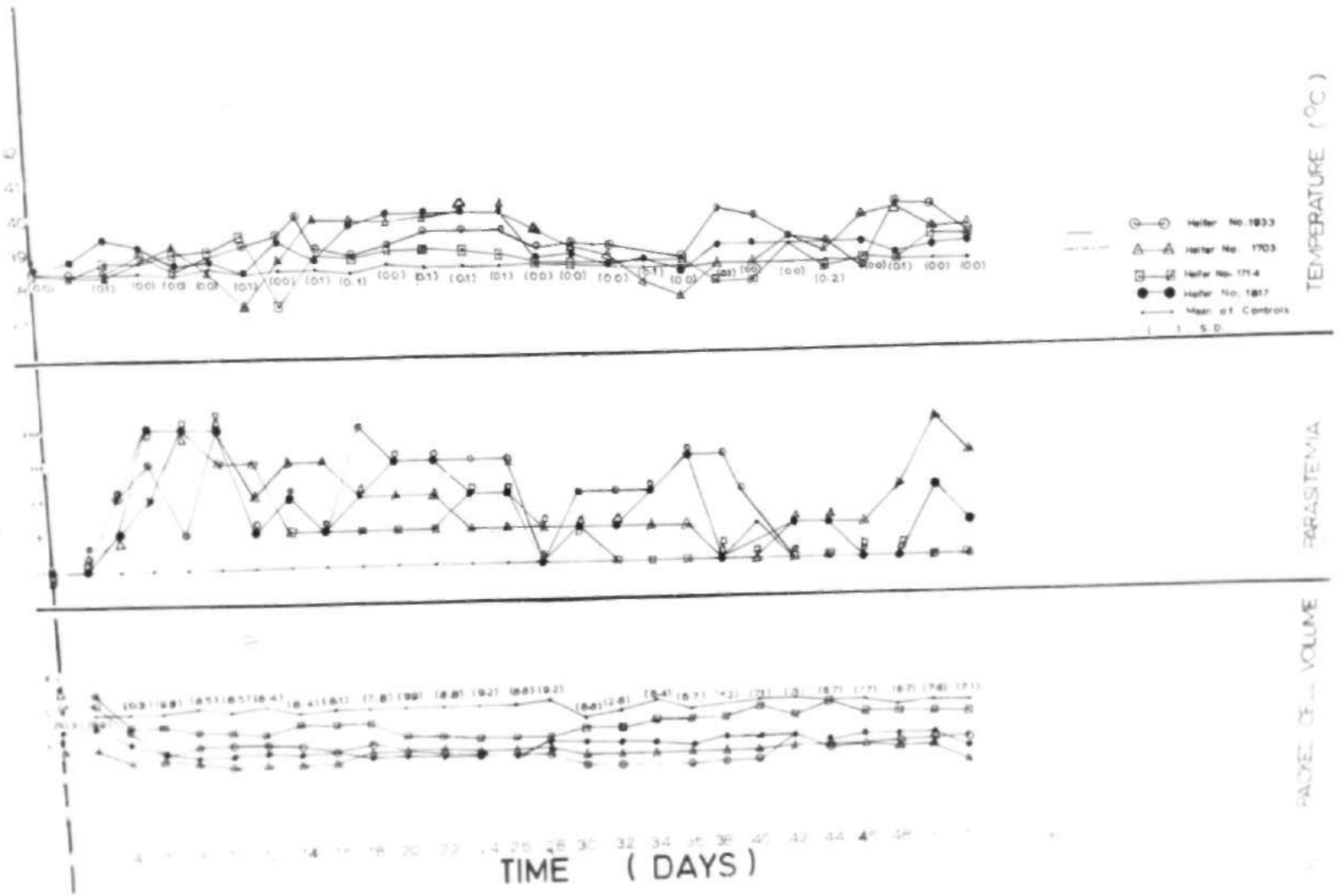
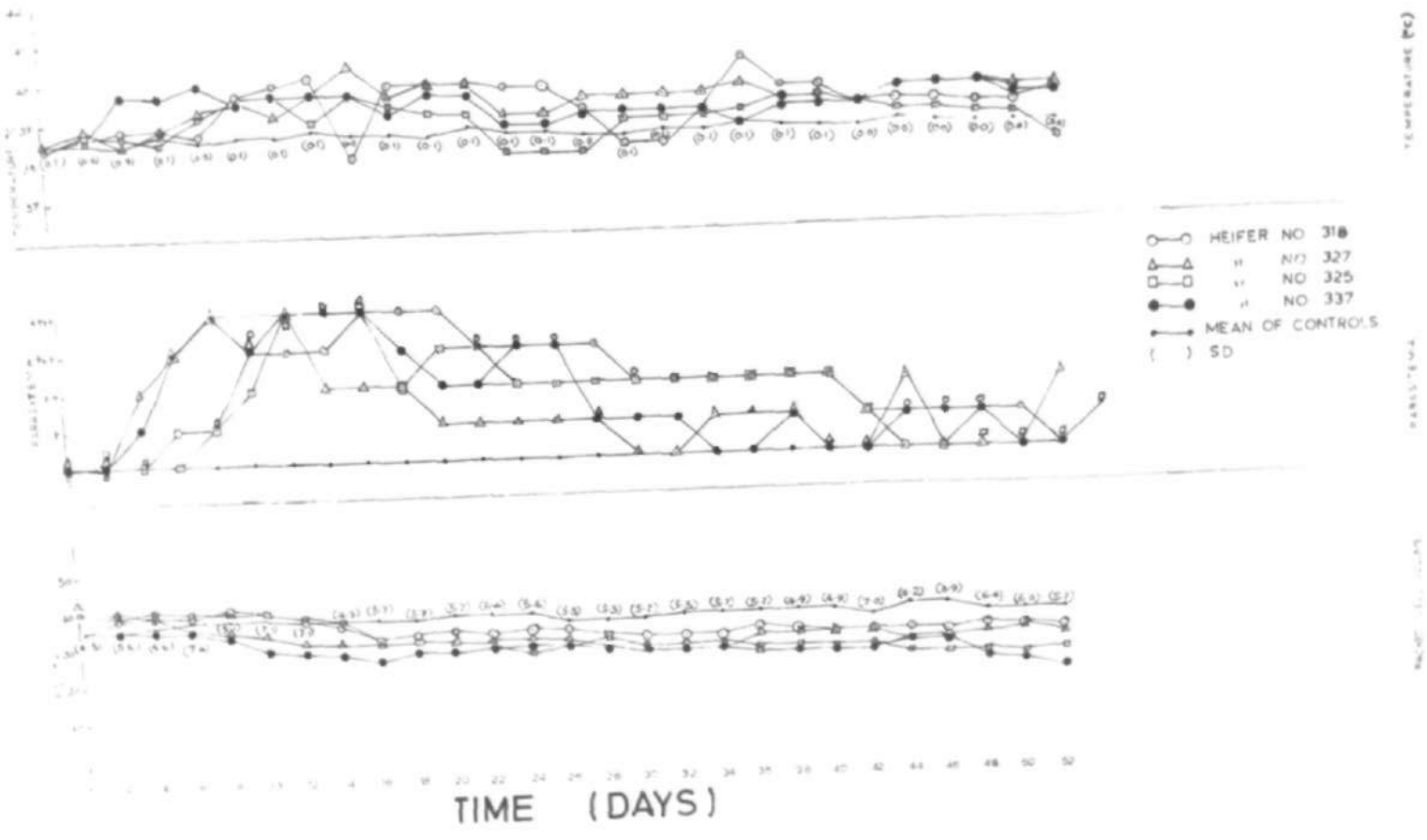

TEMPERATURE (°C)
Heifer No. 1933
Heifer No. 1703
Heifer No. 1714
Heifer No. 1817
Mean of Controls
( ) S.D.
PACKED CELL VOLUME
PARASITEMIA
TIME ( DAYS )

Figure 2

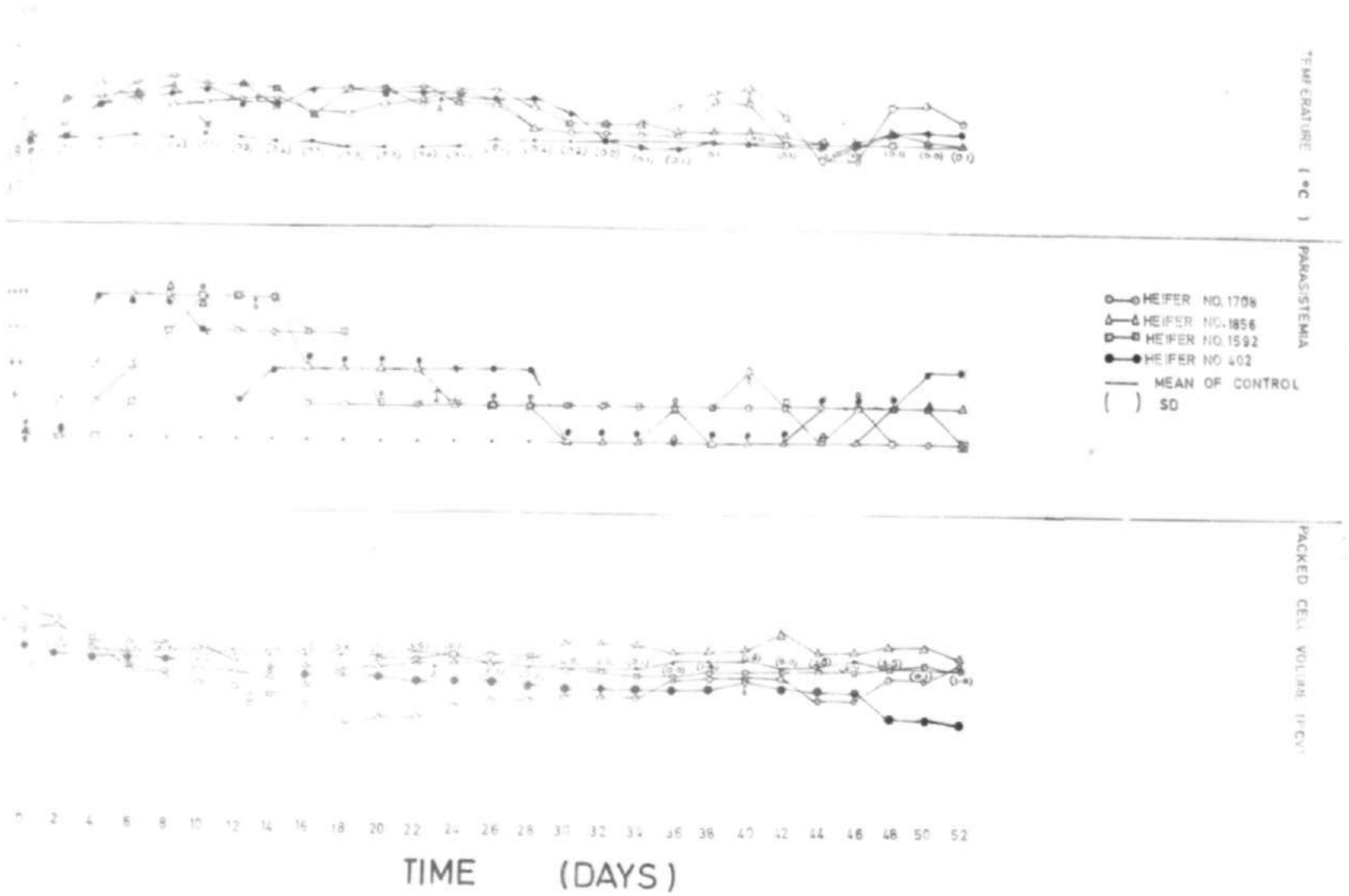
Temperature curves parasitemia and packed cell volume (PCV) in Zebu heifers infected with Trypanosoma vivax during the second trimester of pregnancy.


HEIFER NO 318
" NO 327
" NO 325
" NO 337
MEAN OF CONTROLS
( ) SD
TIME (DAYS)

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Figure 3

Temperature curve, parasitemia and packed cell volume (PCV) in Zebu heifers infected with Trypanosoma vivax during the third trimester of pregnancy.


TEMPERATURE ( °C )
PARASITEMIA
HEIFER NO. 1709
HEIFER NO. 1856
HEIFER NO. 1592
HEIFER NO. 402
MEAN OF CONTROL
( ) SD
PACKED CELL VOLUME (PCV)
0 2 4 6 8 10 12 14 16 18 20 22 24 26 28 30 32 34 36 38 40 42 44 46 48 50 52
TIME (DAYS)

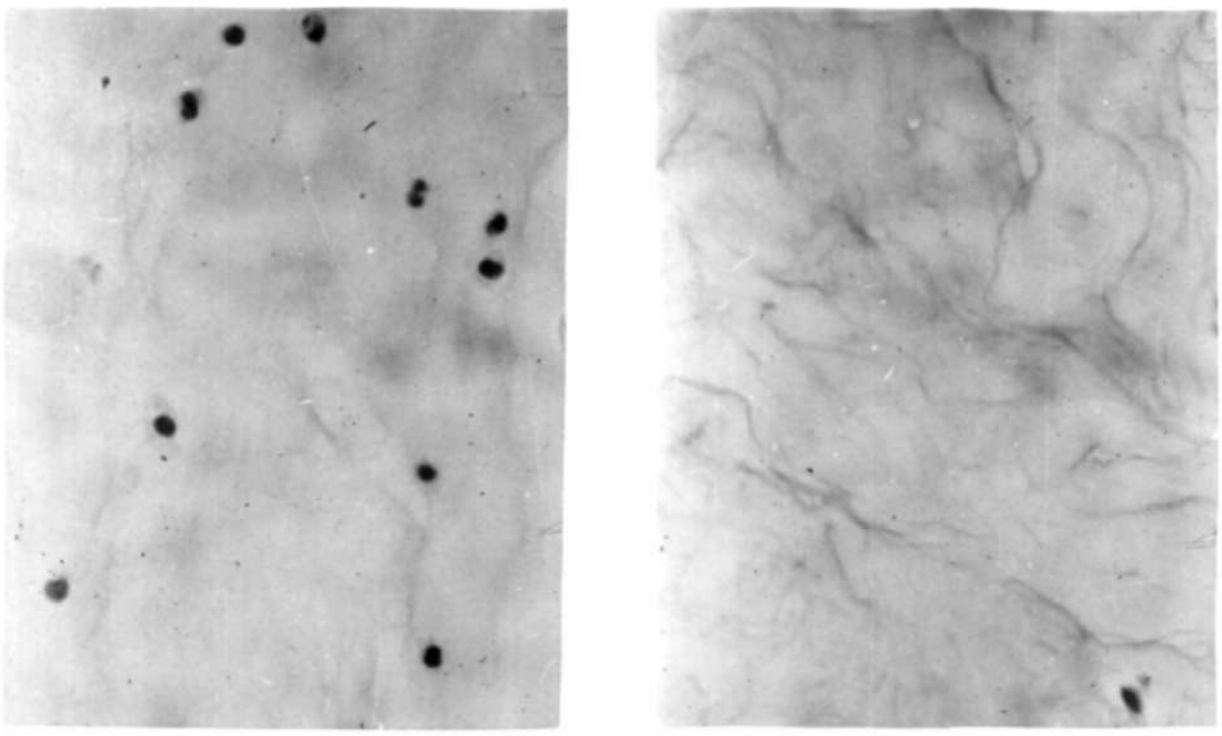
135

Figure 4:

Photomicrograph of a section of the fetal membrane of a heifer infected during the third trimester. Note the mononuclear cells (x 1000).

5: Photomicrograph of a section of the fetal membrane of a control heifer for the third trimester trial. (x 1000).

6: Gross photograph of the vulva opened to show edema in a heifer infected during the first trimester of pregnancy. (x 0.5).



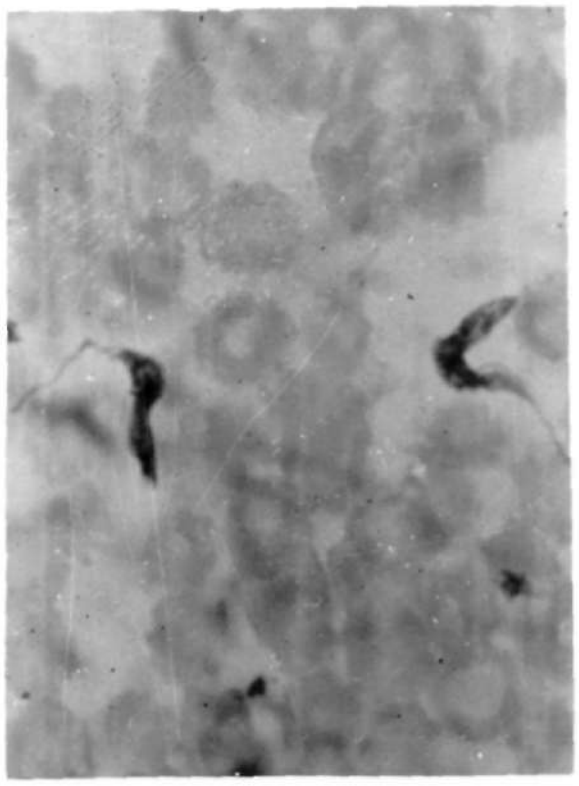


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Figure 7: Photograph of an emaciated calf pulled out before dam died of T. vivax infection. Calf died within 24 hours after forced extraction (x 0.5).

8: Photograph of a dead premature fetus delivered by a heifer infected during the third trimester of pregnancy (x 0.5).

9: Photomicrograph of a fetal thin blood smear illustrating T. vivax in a premature claf following maternal infection during the third trimester of pregnancy. (x 1000).





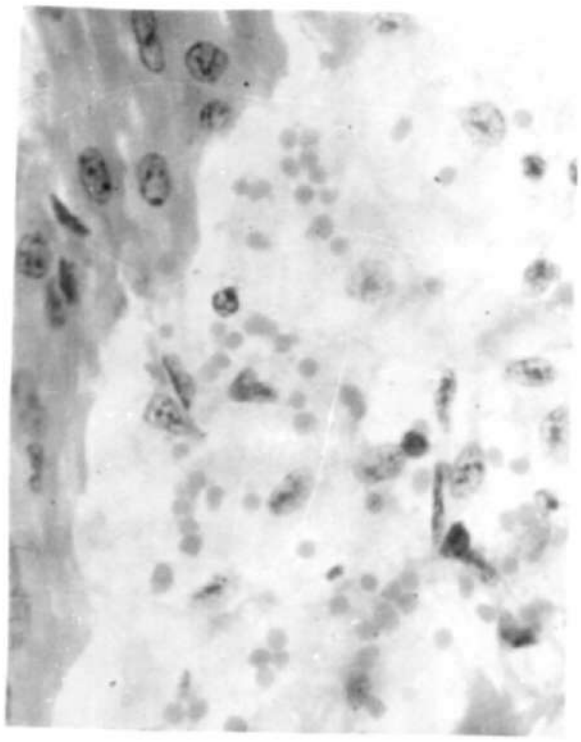


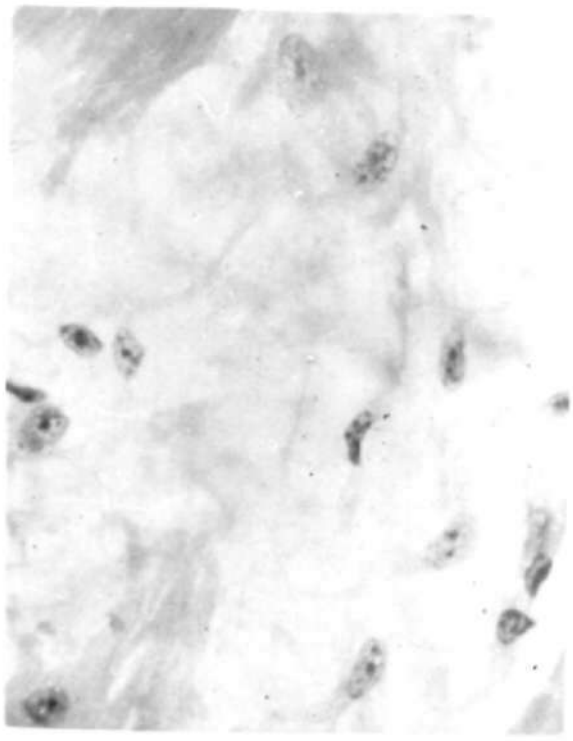
139

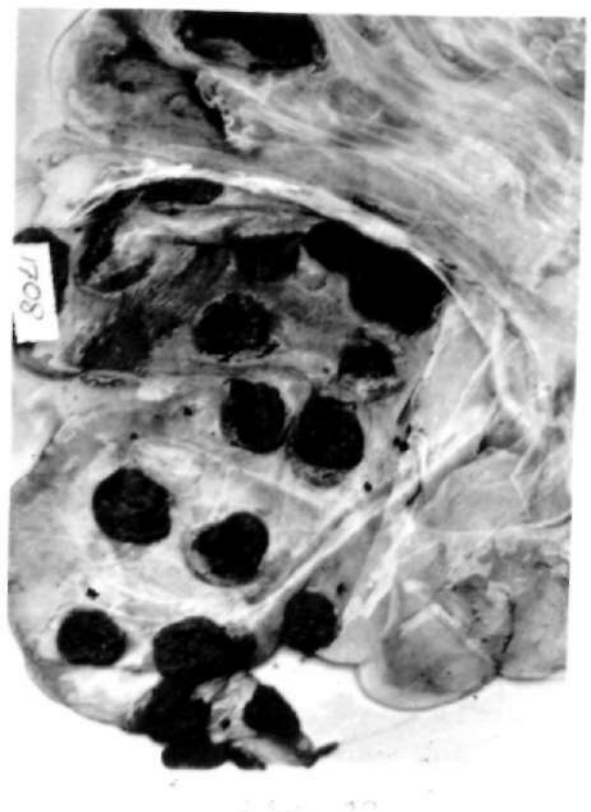
Figure 10: Photomicrograph of a heart section of one of the premature calves. Note the extravasation and edema (x 1000).

11: Photomicrograph of a heart section from one of the premature calves. Note the edema. (x 1000).

12: Photograph of a fetal membrane from an infected heifer (No. 1708) showing no gross lesions (x 0.5).






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# PART IV

## IMMUNOGLOBULIN (IgM and IgG) LEVELS IN BOVINE TRYPANOSOMIASIS

# INTRODUCTION

In mammals, immune response to infection is characterised by a sequential synthesis of different classes of immunoglobulins. The initial immunoglobulin M (IgM) synthesis is replaced by immunoglobulin G (IgG) synthesis (Baner and Stavitsky, 1961; Barett, 1974). In trypanosomiasis, this sequence is modified and the infection is characterised by the prolonged production of IgM antibodies (Masseyeff and Lamy, 1966; Luckins, 1972, 1973, 1976). This disease is not only characterised by this prolonged production of IgM, but also a greatly elevated level of the antibody (Klein et al., 1967; Houba et al., 1969; Rees, 1969; Seed et al., 1969; Baker and Taylor, 1971; Luckins, 1972 and 1976; Kobayashi and Tizard, 1976), rising, on some occasions 20 times the pre-infection level (Kobayashi and Tizard, 1976). In contrast, IgG levels tend to remain constant at values close to normal (Kobayashi and Tizard, 1976) though values 2 - 4 times the preinfection levels have been reported (Rees, 1969; Luckins, 1972). This prolonged synthesis of IgM, though not pathognomonic, has been used as a basis for the diagnosis of trypanosomiasis (Lumsden, 1966; Binz et al., 1968; Goidl and Marsden, 1974).

The tremendous production of IgM seems to be directed against a number of antigens, While some are specifically antitrypanosomes (Seed et al., 1969) others may be heterophil (Houba et al., 1969; Mackenzie et al., 1973) or directed against autoantigens (Mansfield and Kreir, 1972; Mackenzie et al., 1973).

Few observations have been made on the effects of trypanosomiasis on serum immunoglobulin levels in cattle (Luckins, 1972; 1976). Using Zebu cattle from tse-tse fly free areas and allowing them natural challenge Luckins (1972) found that there was a significant increase in IgM and IgG levels. Increases of 2 · 9 fold preinfection levels of IgM were reported two weeks after the cattle were introduced into the enzootic area and these persisted for several months. IgG levels were reported to increase only 2-fold during the period. Similar observations were reported by the same author in 1976 in experimental Trypanosoma congolense and Trypanosoma vivax infections.

This work was carried out to determine the IgM and IgG levels in Trypanosoma vivax infection and how the levels are affected by the different stages of pregnancy in heifers.

# MATERIALS AND METHODS

## Experimental Animals

A group of heifers aged 3 years were acquired from the Kano State Government LIBC farm at Gumel and from NAPRI farm at Shika. The heifers were selected after careful examination which included the examination of the ovaries and genitalia. The animals were transported in a truck to the Faculty and housed in the research pen of the Faculty.

Two days after their arrival at the Faculty, they were dipped using Pfizona$^{R}$ (Pfizer Products Ltd., Ikeja, Nigeria) and dewormed with Thiabendazol$^{R}$ (Thiabenzol, Merck Sharp and Dohme, England) and Ranide$^{R}$ (Merck Sharp and Dohme, England) 2 weeks apart. The animals were screened for brucellosis and leptospirosis. In addition, their blood was evaluated for baseline hematological data and for the presence of blood parasites. The animals that were positive for piroplasms were treated with Imizol$^{R}$ (The Wellcome Foundation Ltd., London). The animals were fed grass hay supplemented with cotton seed cake and wheat offals for 6 months before the experiments and during the experimental period. They were also given mineralised salt licks (Pfizer Products Ltd., Ikeja, Nigeria) and water ad-lib.

At the end of the experiments, the non pregant infected animals were treated with Novidium chloride (Novidium, Major and Co., Nig Ltd , Kano Nigeria) while the pregnant infected animals were treated after calving.

Trypanosomes Used

The Trypanosoma vivax used is designated Y55 and was isolated from a natural infection in N'dama cow (Leeflang et al., 1976).

Stabilate used was preserved in 5% DMSO and stored in liquid nitrogen. Prior to use in these experiments, the stabilates were first inoculated into sheep which subsequently developed clinical trypanosomiasis. Blood from this sheep was then used to infect the experimental heifers

Experimental Design

Four groups of 6 heifers were used for the experiments. In each group, 4 animals were infected while 2 were uninfected controls The first group of animals were non-pregnant animals used for fertility trial - as described in Chapter I. The second, third and fourth groups were those used for studying the effects of experimental infection on first, second and third trimester pregnancy respectively as described in

Chapter III. Each experimental animal was inoculated intravenously (via the jugular vein) with approximately $1.5 \times 10^6$ T. vivax.

The first group of animals, 2 were infected on day 14 and the remaining on day 16 of their estrous cycles while experimental animals in groups 2, 3 and 4 were inoculated on day 60, 4th month and 7th month of their pregnancies respectively.

Following inoculation, the animals (including the controls) were bled daily for the first 2 or 3 weeks and thereafter thrice a week. Serum samples were collected once a week for 3 weeks before the inoculation, on the day of inoculation and once a week thereafter. Serum samples were also collected once a week for 4 weeks after treatment. Blood collections were via the jugular venipuncture into vacutainer tubes with ethylene diaminetetra-acetic acid (EDTA) (1 mg/ml blood) as anticoagulant. Samples for sera were collected in plain vacutainer tubes without anticoagulant. Such samples were allowed to coagulate at room temperature for 4 hours and centrifuged at 1,000R.P.M for 10 minutes at $4^{\circ}C$. The sera were separated out into labelled serum vials and stored at - $20^{\circ}C$ until assayed.

(a) Hematocrit centrifugal test (HCT) for Trypanosomes

A microhematocrit capillary tube containing approximately 70 ul of blood was centrifuge for 5 minutes as for measurement of PCV. The tubes were then placed on a clean glass slide. A drop of immersion oil was put on top of the capillary tubes with the oil filling the spaces between the capillary tubes. The buffycoat - plasma junction was then examined under the microscope using the oil immersion objective as described by Woo (1969). The degree of parasitemia was classified as follows:-

\+ = occasional trypanosomes seen

++ = 1 to 2 trypanosomes seen

+++ = 3 to 20 trypanosomes seen

++++ = Over 20 trypanosomes seen

(b) Thin Blood Film for Trypanosome Identification

Clean, grease-free microscope slides were used. About 5 microlitres of blood were placed at one end of the slide and a thin spread made. The slides were quickly air-dried and fixed for 3 minutes in methanol. After this, the slides were stained in 10% Giemsa for 30 minutes, dried and about 100 fields scanned under the oil immersion (x 790).

(c) Packed Cell Volume (PCV)

In order to estimate the degree of anemia, the packed cell volume (PCV) was determined by the micro-hematocrit method using the international microhematocrit centrifuge.

Reproductive and Pregnancy Examination

A reproductive evaluation of all the animals was carried out on the different farms of purchase to ensure that only cycling animals were selected. Also during the 6 months acclimatization period, reproductive examination of all the heifers was done. The heifers were also rectally examined on day 40 after breeding, on the day of infection and twice monthly thereafter. To achieve this, an obstetrical glove is worn and lubricated with soap. The animals were properly restrained in a chute. The hand was then introduced into the rectum and the cervix identified. The uterus was then retracted by the direct method as described by Zemjanis (1970). The ovaries and the genitalia were then palpated. For pregnancy examination, the same procedure was carried out and the uterus and the entire uterine horns palpated as described by Zemjanis (1970).

Immunoglobuline Level Determination

(a) Immunoglobulin G (IgG) determination:

A single radial immunodiffusion method of Fahey and McKelvey (1965) was used for the quantitative determination of IgG levels in the collected serum samples. The IgG kits were obtained from Miles Laboratories in England) and stored for about 20 days before use in a refrigerator at $4^{o}C$. On the days the analyses were carried out, required number of plates were brought out and allowed to warm up to room temperature. The serum samples to be analysed were also treated similarly.

Preparation of the Standard Curve: 2.5 ul of each reference standard provided were carefully pipetted into the wells on the immunodiffusion plates as indicated with the aid of disposable micropipettes provided in the kit. The required 2.5 ul volume was indicated on the capillary tubes between two black lines. The concentration of the reference standards were 4,000, 2,000, 1,000 and 500 mg/100 ml.

Three drops of water were pipetted into the moisture trough bordering the plate. The cover was replaced and the plate left undisturbed at room temperature for 18 hours.

Precipitin rings were measured directly from the calibrated immunodiffusion plates; each ruling on the plate being one millimetre and indicated on record sheets also provided in the kit. The ring diameter of the standards (X axis) were then plotted against the known concentrations of the standards (Y axis) on the semi-logarithmic graph paper provided.

Sample Analysis: A pretest run of some samples was carried out and the diameter of some found to be larger than the diameter of 4000 mg/100ml reference standard. As a result of this, all the samples were then diluted 1:5 with 0.9% saline solution. The samples were then coded for identification purposes and a 2.5 ul of each diluted sample pipetted into the different wells with a total of 17 samples analysed on one plate. The plates were then treated like the standards and precipitin rings measured directly from the calibrated immunodiffusion plates and recorded. The concentration of IgG in each sample were determined by reading against the standard curve. The final concentration of each sample was then obtained by multiplying by 5, the dilution factor.

(b) Immunoglobulin M (IgM) determination:

The same single radial immunodiffusion method of Fahey and McKelvey (1965) was used for the quantitative determination of IgM levels in the collected serum samples. The IgM kits were obtained from Miles Laboratories in England and stored for about 20 days before use in a refrigerator at 4°C. On the days the analyses were carried out, a required number of plates were brought out and allowed to come to room temperature. The serum samples to be analysed were all allowed to come room temperature.

Preparation of the standard curve: 10 ul of each reference standard provided were carefully pipetted into the wells on the immunodiffusion plates as indicated with the aid of disposable micropipettes also provided in the kit. The required 10 ul volume was indicated on the capillary tubes between two black lines. The concentration of the reference standards were 520, 260, 130 and 60 mg/100ml.

Three drops of water were pipetted into the moisture trough bordering the plate. The cover was replaced and the plates left undisturned at room temperature for 22 hours.

Precipitin rings were measured directly from the calibrated immunodiffusion plate, each ruling on the plate being one millimeter and recorded on record sheets also provided in the kit. The ring diameter of the standard (X axis) was then plotted against the known concentrations of the standards (Y axis) on the semi-logarithmic graph paper provided.

Sample Analysing: As with the IgG determination, a pretest run was also carried out and a 1:5 dilution of all the samples was done with 0.9% saline solution. The samples were then coded for identification and a 10 ul of each diluted sample pipetted into the different wells with a total of 17 samples analysed on each plate.

The plates were then treated like the standards and the precipitin ring diameters measured directly from the calibrated immunodiffusion plates and recorded. The concentration of IgM in each sample was determined by reading against the standard curve. The final concentration of each sample was obtained by multiplying by 5, the dilution factor.

Statistical Analysis

Analysis of variance was performed on the IgM and IgG values of the animals in the different treatment groups using the method of Statistical Package for Social Sciences (SPSS) and the Genstat V method.

Comparisons of values in the different treatment groups were done. In addition, interactions such as group/period interaction, group/animal interaction and period/animal interaction were considered. A 95 percent confidence level was chosen thus where $P>0.05$, it was considered non-significant.

## RESULTS

The standard curves of IgM and IgG obtained by using the provided IgM and IgG are shown in figures 1 and 2. Consistent curves were obtained on the different days on which the analyses were done. The IgM and IgG levels in the different animals within the 4 treatment groups are shown in appendix 5, 6, 7, and 8 while the mean values and the infection differentials for the different groups are shown in tables 1 - 8. The mean values for the control animals in the different groups are also shown in tables 9 - 16. These mean values are summarised in figures 3 - 10.

In all the groups, there was a significant rise in IgM levels starting from the first week post infection (pi) corresponding with the first rise in parasitemia. In all the groups, 7 - 8 fold increases were recorded. Peak levels were found between the fourth and nineth week p.i. However, there were differences in the rate of increases between the groups ($P<0.001$) and in the ability of the different groups to maintain peak leavels ($P<0.001$). Among animals within the same group there were differences in the IgM levels but the differences were statistically non-significant ($P>0.05$). When the interclass correlation coefficient (interaction) analyses were done, it

was found that the rise over the stated period between the groups was statistically significant ($P<0.05$). Thus the group/period interaction was significant. Also both group/animal interaction and period/animal interaction were statistically significant (see analysis of variance table for IgM).

In the non-pregnant heifers the IgM rise was gradual and peak levels of over 2,000 mg/100ml - 2,412.5 mg/100ml were attained from the 4th week. In the heifers infected during the first trimester of pregnancy the rise in IgM levels was more rapid than those in the non-pregnant group. By the third week, levels over 2,000 mg/100 ml were already recorded and the peak levels of IgM were more sustained (see figure 4) and over - 8-fold increases were measured.

In heifers infected during the second trimester, the IgM response was even more rapid than those infected in first trimester. In this group of heifers, IgM levels of 2,400 mg/100ml were recorded in the third week and response was sustained for over 14 weeks. In heifers infected during the third trimester, it was found that the IgM response was gradual, comparable only to those of non-pregnant heifers. Here, only 4-5 fold increases were recorded and the IgM response was less sustained than those of other pregnant groups.

In all the groups the difference between the infected animals and the controls was highly significant ($P<0.001$). No statistically significant difference was however found in the controls in the different groups although heifers in their third trimester pregnancy had higher levels of IgM.

The IgG results showed similar pattern of increases. In all the treatment groups the rise was gradual and the IgG response comes after the IgM response. In all the groups a 1.2-2.5 fold increases in IgG levels were recorded. Unlike the IgM response where peak levels were recorded about the 3rd - 4th week, peak levels of IgG were recorded from the 6th - 7th week except in the second trimester heifers where peak levels were recorded from the 4th week.

The differences in the IgG response in heifers in the different groups were highly significant ($P<0.001$). Unlike in the case of IgM response, differences amongst heifers in the groups were also significant ($P<0.05$). The IgG response of the different groups over the 15 week period (Group/Period Interaction) was significantly different ($P<0.05$). Similarly, the group/animal and period/animal interactions were also statistically significant (see analysis of variance table for IgG)

In the non-pregnant heifers, mean peak levels of 5,650 mg/100 ml were recorded. In the individual heifers, peak levels ranged from 4,600 mg/100ml in heifer L31 to 7,000 mg/100ml in heifer L32 giving a 2 - 2.8 fold increase. The mean IgG levels in the control heifers ranged from 2,500 mg/100ml -2,775.0mg/100ml. No significant increases were recorded throughout the period of investigation in the control heifers.

In heifers infected during the first trimester, mean peak levels of 4,837 - 5,875mg/100ml were recorded. The peak levels in the individual heifers ranged from 5,500 - 7,000mg/100ml giving a 2.2 - fold increase. In the control heifers in this groups, the mean IgG level ranged from 3,050 - 3,750 mg/100ml. Variation in the levels of IgG in this control group over the experimental period was not significant. The IgG levels in the infected heifers were significantly higher than those of the non-infected controls ($P<0.001$). Figure 5 shows that the mean peak IgG levels the infected heifers were attained 6 week p.i. and the response was sustained throughout the investigation period with only slight drop during 12th and 13th week.

In heifers infected during the second trimester, there was a rapid IgG response reaching a peak at about the 4th week p.i. The mean peak level was not well sustained in this group. There was a gradual decrease initially and from the 11th week the decrease was rapid falling almost to the preinfection level by the 15th week. The peak levels in the individual heifers infected ranged from 5,500 - 7,000 ng/100ml. Intragroup differences between infected animals were significant ($P<0.05$). In this group also a 2.2 - 2.8 fold increase was recorded for the IgG levels. Variation in the IgG levels in the control heifers was non-significant ($P>0.05$) though some increases were recorded from the 9th week p.i. The IgG levels in the infected heifers were significantly higher than those of the control ($P<0.001$).

In heifers infected during the third trimester, there was a gradual IgG response to the infection. Optimum or mean peak levels were attained from the 6th week p.i and sustained. However, in this group, the ability to respond to the infection was less as 1.2 -1.4 fold increase was recorded on the overall. Between the 6th and 9th week, the peak response was maintained after which, there was a gradual drop in the levels of IgG. Between the 9th and 14th week, variations in the levels of IgG existed between the animals. The peak levels of IgG

in the infected heifers in the group ranged from 4,600 - 5,500 giving 1.2 - 1.4 fold increase. The IgG levels in the control heifers which were high when compared to those of other groups decreased significantly from the 7th week. This decrease was uniform in the 2 control animals.

# DISCUSSION

The results showed that a single T. vivax infection of these heifers produced a tremendous increase in the IGM level and lesser increases in the IgG level. Significant increases in IgM levels coincided with the first wave of parasitemia which occurred 7 - 14 days p.i. These results are in conformity with those reported by Luckins (1972, 1973 and 1976) in Zebu cattle and those of Binz et al., (1968). Mattern (1962, 1964 and 1968), Lumsden (1966) Masseyeff and Lamy (1966) in humans. The IgG levels in the infected heifers showed a 1.5 - 1.5 fold increase. This also agrees with the findings of Masseyeff and Lamy (1966) Masseyeff et al., 1972 and Luckins (1972, and 1976). However, though there is general agreement on the changes in the levels of IgM in trypanosomiasis the findings in the case of IgG differ. In trypanosomiasis, Masseyeff and Lamy (1966) and Luckins (1972 and 1976) found increased levels of both IgM and IgG in human and bovine cases. Similarly Frommel et al., (1970) found increased levels of IgG in rabbits infected with T. equiperdium but Mouba et al., (1969) found no such increases in IgG levels in monkeys infected with T. gambiense, T. rhodesiense and T. brucei. Clarkson and Penhale (1975) found that IgG levels in cattle infected experimentally with T. vivax did not

increase until 50 days after infection. It is thought that finding an elevated level of IgG in clinical trypanosomiasis depends on the sensitivity of the method(s) used and on whether the subject is pregnant or not and if pregnant, the stage of the pregnancy.

From the result reported here it appears that in bovine trypanosomiasis, there is a general IgM increase in all infected animals which is sustained by pregnancy and the stage of pregnancy. Among the pregnant heifers it can also be seen that animals infected during the third trimester have a poorer ability to maintain the elevated levels of IgM than those in their first and second trimester stages of pregnancy. In heifer No. 402, there was a gradual decrease in IgM levels until it died of the infection.

The IgG response in the infected heifers though slight (1.2 - 2.5 compared to 8 fold increase in IgM) showed rather interesting pattern. Whereas IgM levels were greatly elevated within the first 14 days p.i., IgG levels during the corresponding period remained low and except in heifers in their second trimester, optimum response was not recorded in all the others until about 42 days p.i. Another interesting aspect of the IgG response was greater group and intragroup variations.

Here again it can be said that generally pregnant heifers responded better than non-pregnant ones apart from heifers infected during the third trimester. In this group of heifers, the initial IgG levels before infection were higher than those of other groups.

If the elevated IgG and IgM levels were a measure of the ability of infected heifers to combat the infection then it could be said that pregnant heifers are more protected than non-pregnant heifers. In addition one could also say that heifers in the third trimester, probably because of the stress of advanced pregnancy are more likely to succumb to trypanosome infection. Our observation is that this is really the true situation. Non-pregnant heifers were more severely affected by the T. vivax infection than heifers infected during the first and second trimester. Heifers in the third trimester were very badly affected (see Part III). The situation during the third trimester may be due to the stress of pregnancy. Since advanced pregnancy is so much stress to the animal, infections such as trypanosomiasis often result in fatality in this group of animals. Similar observations have been made with other infections such as tuberculosis (Abdulkadir, personal communication).

One point of interest though is that it is not clear if the elevated IgM response is protective or not. Luckins (1976) reported that protective antibodies against antigenic variants of T. congolense were found in the IgM fractions of sera though the protective activity of whole serum was greater than that of the IgM fraction alone. Similarly, protective activity has been found in the IgM fraction of sera from rabbits infected with T. brucei, T. gambiense and T. equiperdium (Seed et al., 1969 and Seed, 1972). Little changes were seen in the protective activity of IgG fractions in T. congolense infected cattle (Luckins, 1976) but according to Seed (1972) protective activity is found in IgG fraction of sera later in infection when the activity of IgM antibodies has declined. In this regard therefore my earlier hypothesis that pregnant animals are more protected against T. vivax infection as shown by their IgM and IgG response is valid.

The relevence of this study is that it has validated the fact that the demonstration of elevated IgM levels can be used as a screening test for the diagnosis of T. vivax infection in cattle. In humans, Maltern (1962, 1964 and 1968) has already demonstrated this. Another important aspect of the work is that it has shown that the reproductive status of the animal should be taken into consideration in the interpretation of the screening test based on elevated IgM levels.

# S U M M A R Y

Immunoglobulin M and G responses in bovine trypanosomiasis (T. vivax infection) were studied in non-pregnant, first, second and third trimester pregnant heifers. In each category, 4 heifers were infected while 2 uninfected were used as controls. IgM and IgG levels in serum samples from the heifers were monitored over a 15 week study period using kits obtained from Miles Laboratories, England.

All the infected heifers showed significant rise in IgM levels when compared to the uninfected controls. A 7 to 8-fold increases were recorded. There were, however differences in the rate of increases and ability to maintain peak level responses ($P<0.01$) between the groups. Infected pregnant heifers in the first and second trimesters responded better than infected non-pregnant and infected heifers in the third trimester of pregnancy. In all the groups, the differences between the infected and control heifers were highly significant ($P<0.001$).

The IgG results showed similar though gradual pattern of increases in the infected heifers. In the different groups, a 1.2 to 2.5-fold increases were recorded. These were significantly different from levels

in the control heifers ($P<0.01$). Again pregnant heifers in the first and second trimesters responded better than non-pregnant and third trimester pregnant heifers.

The results correlated with the observation that given the same conditions, clinical manifestations of T. vivax infection is more severe in non-pregnant than in pregnant heifers except those in advanced pregnancy. It is suggested that in using elevated IgM levels as a screening test for the diagnosis of T. vivax infection, consideration should be given to the reproductive status of the animal in the interpretation of the test.

Table 1: Mean IgM levels (± S.D.) of infected animals and their corresponding mean infection differential △IgM, ± S.D. non-pregnant group

| Week | Mean (± S.D) mg/100ml | Mean△ IgM (± S.D.) |
|---|---|---|
| -2 | 300 ± 0.0 | 62.5 ± 0 |
| -1 | 300 ± 0.0 | 0.0 ± 0 |
| 0 | 300 ± 0.0 | 62.5 ± 0 |
| 1 | 862.5 ± 505 | 337.5 ± 441 |
| 2 | 1531.3 ± 604 | 1218.8 ± 663 |
| 3 | 2065.5 ± 791 | 1718.0 ± 839 |
| 4 | 2181.3 ± 839 | 1811.3 ± 838 |
| 5 | 2412.5 ± 375 | 2112.5 ± 375 |
| 6 | 2412.5 ± 375 | 2112.5 ± 375 |
| 7 | 2412.5 ± 375 | 2065.6 ± 344 |
| 8 | 2412.5 ± 375 | 2003.0 ± 344 |
| 9 | 2412.5 ± 375 | 2081.3 ± 438 |
| 10 | 2255.0 ± 433 | 1925.0 ± 433 |
| 11 | 2012.5 ± 325 | 1737.5 ± 375 |
| 12 | 1850.0 ± 0.0 | 1550.0 ± 0 |
| 13 | 1712.5 ± 275 | 1412.5 ± 275 |
| 14 | 1343.8 ± 380 | 1043.8 ± 380 |
| 15 | 1206.3 ± 188 | 906.3 ± 188 |

Table 2: Mean IgM levels (± S.D.) of infected animals and their corresponding mean infection differential, △ IgM (± S.D) - first Trimester group

| Week | Mean (± S.D.) (mg/100ml) | Mean △ IgM (± S.D) |
|---|---|---|
| -2 | 351.3 ± 62.5 | 41.6 ± 72.2 |
| -1 | 351.3 ± 62.5 | 41.6 ± 72.2 |
| 0 | 351.3 ± 62.5 | 41.6 ± 72.2 |
| 1 | 621.5 ± 507 | 596.9 ± 478 |
| 2 | 1781.3 ± 985 | 1481.3 ± 985 |
| 3 | 2181.3 ± 938 | 1881.3 ± 838 |
| 4 | 1950.0 ± 751 | 1650.0 ± 751 |
| 5 | 2275.0 ± 650 | 1975.0 ± 650 |
| 6 | 2600.0 ± 0.0 | 2300.0 ± 0.0 |
| 7 | 2600.0 ± 0.0 | 2300.0 ± 0.0 |
| 8 | 2600.0 ± 0.0 | 2300.0 ± 0.0 |
| 9 | 2600.0 ± 0.0 | 2300.0 ± 0.0 |
| 10 | 2600.0 ± 0.0 | 2300.0 ± 0.0 |
| 11 | 2600.0 ± 0.0 | 2300.0 ± 0.0. |
| 12 | 2275.0 ± 650 | 1975.0 ± 650 |
| 13 | 2087.5 ± 633 | 1787.5 ± 653 |
| 14 | 2412.5 ± 375 | 2112.5 ± 375 |
| 15 | 2412.5 ± 375 | 2112.5 ± 375 |

Table 3: Mean IgM levels (± S.D.) of infected animals and their corresponding mean infection differential, △ IgM (± S.D.) - Second trimester group

| Week | Mean (± S.D.) (mg/100ml) | Mean △ IgM (± S.D) |
|---|---|---|
| -2 | 393.8 ± 63 | 62.4 ± 2.0 |
| -1 | 362.2 ± 72 | 0.0 ± 102 |
| 0 | 362.5 ± 72 | 62.5 ± 72 |
| 1 | 443.8 ± 134 | 81.3 ± 134 |
| 2 | 1387.5 ± 534 | 1078.3 ± 258 |
| 3 | 2412.5 ± 375 | 2050.0 ± 375 |
| 4 | 2600.0 ± 0.0 | 2253.1 ± 31 |
| 5 | 2600.0 ± 0.0 | 2300.0 ± 0 |
| 6 | 2600.0 ± 0.0 | 2284.4 ± 31 |
| 7 | 2600.0 ± 0.0 | 2237.5 ± 0 |
| 8 | 2412.5 ± 375 | 2050.0 ± 375 |
| 9 | 2412.5 ± 375 | 2050.0 ± 375 |
| 10 | 2225.0 ± 433 | 2050.0 ± 375 |
| 11 | 2412.5 ± 375 | 2050.0 ± 375 |
| 12 | 2412.5 ± 375 | 1987.5 ± 375 |
| 13 | 2225.0 ± 433 | 1800.0 ± 433 |
| 14 | 2225.0 ± 433 | 1800.0 ± 433 |
| 15 | 1762.5 ± 616 | 1200.8 ± 650 |

Table 4: Mean levels of IgM in infected heifer (± S.D.) and their corresponding mean infection differential △ IgM (± S.D.) - third trimester group

| Week | Mean (± S.D.) (mg/100ml) | Mean △ IgM (± S.D.) |
|---|---|---|
| -2 | 575 ± 100 | 268.8 ± 338 |
| -1 | 618.8 ± 256 | 206.3 ± 142 |
| 0 | 650.0 ± 206 | 175.0 ± 150 |
| 1 | 962.5 ± 390 | 437.5 ± 390 |
| 2 | 1625.0 ± 650 | 1231.3 ± 615 |
| 3 | 1950.0 ± 751 | 1425.0 ± 751 |
| 4 | 2600.0 ± 0.0 | 2075.0 ± 0 |
| 5 | 2600.0 ± 0.0 | 2075.0 ± 0 |
| 6 | 2600.0 ± 0,0 | 2075.0 ± 0 |
| 7 | 2600.0 ± 0.0 | 2975.0 ± 0 |
| 8 | 2412.5 ± 375 | 1887.5 ± 375 |
| 9 | 2412.5 ± 375 | 1887.5 ± 375 |
| 10 | 2275.0 ± 650 | 1750.0 ± 650 |
| 11 | 2275.0 ± 650 | 1750.0 ± 650 |
| 12 | 1918.6 ± 932 | 1493.0 ± 932 |
| 13 | 1777.5 ± 1198 | 1535.0 ± 856 |
| 14 | 1452.5 ± 1070 | 1235.0 ± 740 |
| 15 | 1916.7 ± 653 | 1391.7 ± 653 |

* Based on reading from three animals, the fourth having died of the diseases.

Table 5: Mean IgG levels (+ S.D) of infected animals and their corresponding mean infection differential (± S.D) - non pregnant group

| Week | Mean (± S.D) mg/100ml) | Mean △IgG (± S.D.) |
|---|---|---|
| -2 | 2637.5 ± 275 | 275.0 ± 0 |
| -1 | 2637.5 ± 275 | 137.0 ± 275 |
| 0 | 2637.5 ± 275 | 137.0 ± 275 |
| 1 | 2775.0 ± 318 | 275.0 ± 318 |
| 2 | 3400.0 ± 404 | 900.0 ± 404 |
| 3 | 3837.5 ± 1156 | 1337.0 ± 1156 |
| 4 | 4175.0 ± 490 | 1400.0 ± 490 |
| 5 | 4175.0 ± 490 | 1675.0 ± 490 |
| 6 | 5050.0 ± 520 | 2775.0 ± 450 |
| 7 | 5650.0 ± 995 | 3150.0 ± 995 |
| 8 | 5650.0 ± 995 | 3150.0 ± 995 |
| 9 | 4837.5 ± 832 | 2087.5 ± 832 |
| 10 | 4837.5 ± 840 | 2337.5 ± 840 |
| 11 | 4837.5 ± 840 | 2337.5 ± 840 |
| 12 | 4437.5 ± 1018 | 1937.5 ± 1018 |
| 13 | 4225.0 ± 1060 | 1725.0 ± 1060 |
| 14 | 4012.5 ± 1045 | 1512.5 ± 1045 |
| 15 | 4012.5 ± 1045 | 1237.5 ± 1045 |

* N = 4

Table 6: Mean IgG levels (± S.D) of infected animals and their corresponding mean infection differential, △ IgG (± S.D.)- first trimester group

| Week | Mean (± S.D.) (mg/100ml) | Mean △ IgG (± S.D.) |
|---|---|---|
| -2 | 3125.0 ± 722 | 625.0 ± 318 |
| -1 | 2950.0 ± 59 | 625.0 ± 318 |
| 0 | 3125.0 ± 722 | 625.0 ± 318 |
| 1 | 3475.0 ± 908 | 700.0 ± 422 |
| 2 | 4212.5 ± 775 | 987.5 ± 425 |
| 3 | 4437.5 ± 1018 | 1000.0 ± 836 |
| 4 | 4837.5 ± 840 | 1437.0 ± 840 |
| 5 | 4837.5 ± 840 | 1437.0 ± 840 |
| 6 | 5875.0 ± 750 | 2825.0 ± 750 |
| 7 | 5875.0 ± 750 | 2475.0 ± 750 |
| 8 | 5875.0 ± 750 | 2475.0 ± 750 |
| 9 | 5875.0 ± 750 | 2475.0 ± 750 |
| 10 | 5875.0 ± 750 | 2750.0 ± 750 |
| 11 | 5875.0 ± 750 | 2475.0 ± 750 |
| 12 | 5437.5 ± 1329 | 2037.5 ± 1329 |
| 13 | 5212.5 ± 1389 | 1812.5 ± 1329 |
| 14 | 5650.0 ± 995 | 1900.0 ± 995 |
| 15 | 5425.0 ± 1132 | 1675.0 ± 1132 |

Table 7: Mean IgG levels in infected heifer (± S.D.) and their corresponding infection differential △ IgG, (± S.D) - second trimester group

| Week | Mean IgG (± S.D.) (mg/100ml) | Mean △ IgG (± S.D.) |
|---|---|---|
| -2 | 2775.0 ± 318 | 275.0 ± 318 |
| -1 | 2775.0 ± 318 | 275.0 ± 318 |
| 0 | 2637.5 ± 275 | 1325.5 ± 275 |
| 1 | 3300.0 ± 905 | 1325.0 ± 895 |
| 2 | 4600.0 ± 0.0 | 2100.0 ± 0.0 |
| 3 | 5275.0 ± 450 | 2775.0 ± 450 |
| 4 | 5500.0 ± 0.0 | 3000.0 ± 0.0 |
| 5 | 5500.0 ± 0.0 | 2725.0 ± 0.0 |
| 6 | 5875.0 ± 750 | 3375.0 ± 750 |
| 7 | 5650.0 ± 995 | 3150.0 ± 995 |
| 8 | 5275.0 ± 450 | 2775.0 ± 450 |
| 9 | 5275.0 ± 450 | 2500.0 ± 450 |
| 10 | 5050.0 ± 520 | 2275.0 ± 520 |
| 11 | 5050.0 ± 520 | 2275.0 ± 520 |
| 12 | 4825.0 ± 450 | 2050.0 ± 450 |
| 13 | 4387.5 ± 425 | 1612.0 ± 425 |
| 14 | 3612.5 ± 736 | 637.0 ± 736 |
| 15 | 3225.0 ± 350 | 450.0 ± 350 |

Table 8: Mean IgG levels in infected animals (± S.D.) and their corresponding mean infection differential IgG (± S.D.) - third trimester group

| Week | Mean (± S.D.) mg/100ml | Mean △ IgG (± S.D.) |
|---|---|---|
| -2 | 3750.0 ± 0.0 | 0.0 ± 0.0 |
| -1 | 3750.0 ± 0.0 | 0.0 ± 0.0 |
| 0 | 3750.0 ± 0.0 | 0.0 ± 0.0 |
| 1 | 3962.5 ± 425 | 212.5 ± 425 |
| 2 | 4387.5 ± 425 | 812.5 ± 350 |
| 3 | 4387.5 ± 425 | 987.5 ± 425 |
| 4 | 4612.5 ± 715 | 862.5 ± 715 |
| 5 | 4612.5 ± 715 | 862.5 ± 715 |
| 6 | 5275.0 ± 450 | 1875.0 ± 450 |
| 7 | 5275.0 ± 450 | 1875.0 ± 450 |
| 8 | 5275.0 ± 450 | 2225.0 ± 450 |
| 9 | 5275.0 ± 450 | 2225.0 ± 450 |
| 10 | 5062.5 ± 875 | 2012.5 ± 875 |
| 11 | 4450.0 ± 1246 | 1212.5 ± 1025 |
| 12 | 4887.5 ± 1225 | 1837.5 ± 1225 |
| 13 | 4312.5 ± 1463 | 1537.5 ± 1055 |
| 14 | 4106.3 ± 1819 | 1743.8 ± 861 |
| 15 | 4916.7 ± 1010 | 1866.7 ± 1010 |

Table 9: Mean IgM levels and standard deviations for the non pregnant controls

| Week | Mean (± S.D.) |
|---|---|
| -2 | 362.5 ± 88.4 |
| -1 | 300.0 ± 0.0 |
| 0 | 362.5 ± 88.4 |
| 1 | 300.0 ± 0.0 |
| 2 | 300.0 ± 0.0 |
| 3 | 462.5 ± 229.8 |
| 4 | 300.0 ± 0.0 |
| 5 | 300.0 ± 0.0 |
| 6 | 300.0 ± 0.0 |
| 7 | 362.5 ± 88 |
| 8 | 425.0 ± 0.0 |
| 9 | 300.0 ± 0.0 |
| 10 | 300.0 ± 0.0 |
| 11 | 300.0 ± 0.0 |
| 12 | 300.0 ± 0.0 |
| 13 | 300.0 ± 0.0 |
| 14 | 300.0 ± 0.0 |
| 15 | 300.0 ± 0.0 |

Table 10: Mean IgM levels and standard deviations for the first trimester control heifers

| Week | Mean (± S.D.) |
|---|---|
| -2 | 300.0 ± 0.0 |
| -1 | 300,0 ± 0.0 |
| 0 | 300,0 ± 0.0 |
| 1 | 362,5 ± 0.0 |
| 2 | 300.0 ± 0,0 |
| 3 | 300.0 ± 0.0 |
| 4 | 300.0 ± 0.0 |
| 5 | 300.0 ± 0.0 |
| 6 | 300.0 ± 0.0 |
| 7 | 300.0 ± 0.0 |
| 8 | 300.0 ± 0.0 |
| 9 | 300.0 ± 0.0 |
| 10 | 300.0 ± 0.0 |
| 11 | 300.0 ± 0.0 |
| 12 | 300.0 ± 0.0 |
| 13 | 300.0 ± 0.0 |
| 14 | 300.0 ± 0.0 |
| 15 | 300.0 ± 0,0 |

Table 11: Mean IgM levels and standard deviations for the second trimester control heifers

| Week | Mean (± S.D.) |
|---|---|
| -2 | 362.5 ± 88.4 |
| -1 | 425.0 ± 0.0 |
| 0 | 300.0 ± 0.0 |
| 1 | 362.5 ± 88.4 |
| 2 | 362.5 ± 88.4 |
| 3 | 362.5 ± 88.4 |
| 4 | 362.5 ± 88.4 |
| 5 | 300.0 ± 0.0 |
| 6 | 300.0 ± 0.0 |
| 7 | 362.5 ± 88.4 |
| 8 | 362.5 ± 88.4 |
| 9 | 362.5 ± 88.4 |
| 10 | 362.5 ± 88.4 |
| 11 | 362.5 ± 88.4 |
| 12 | 425.0 ± 0.0 |
| 13 | 425.0 ± 0.0 |
| 14 | 425.0 ± 0.0 |
| 15 | 425.0 ± 0.0 |
| 16 | 425.0 ± 0.0 |

Table 12: Mean IgM levels and standard deviations for the third trimester control heifers

| Week | Mean (± S.D.) |
|---|---|
| -2 | 525.0 ± 141 |
| -1 | 525.0 ± 141 |
| 0 | 525.0 ± 141 |
| 1 | 525.0 ± 141 |
| 2 | 525.0 ± 141 |
| 3 | 525.0 ± 141 |
| 4 | 525.0 ± 141 |
| 5 | 525.0 ± 141 |
| 6 | 525.0 ± 141 |
| 7 | 525.0 ± 141 |
| 8 | 525.0 ± 141 |
| 9 | 525.0 ± 141 |
| 10 | 525.0 ± 141 |
| 11 | 525.0 ± 141 |
| 12 | 425.0 ± 0.0 |
| 13 | 425.0 ± 0.0 |
| 14 | 425.0 ± 0.0 |
| 15 | 525.0 ± 141 |

Table 13: Mean IgG levels and standard deviations for the non-pregnant controls

| Week | Mean (± S.D.) |
|---|---|
| -2 | 2775.0 ± 388.9 |
| -1 | 2500.0 ± 0.0 |
| 0 | 2500.0 ± 0.0 |
| 1 | 2500.0 ± 0.0 |
| 2 | 2500.0 ± 0.0 |
| 3 | 2500.0 ± 0.0 |
| 4 | 2775.0 ± 388.9 |
| 5 | 2500.0 ± 0.0 |
| 6 | 2500.0 ± 0.0 |
| 7 | 2500.0 ± 0.0 |
| 8 | 2500.0 ± 0.0 |
| 9 | 2775.0 ± 388.9 |
| 10 | 2500.0 ± 0.0 |
| 11 | 2500.0 ± 0.0 |
| 12 | 2500.0 ± 0.0 |
| 13 | 2500.0 ± 0.0 |
| 14 | 2500.0 ± 0.0 |
| 15 | 2775.0 ± 388.9 |

Table 14. Mean IgG levels and standard deviations for first trimester control heifers

| Week | Mean (± S.D) |
|---|---|
| -2 | 3400.0 ± 495 |
| -1 | 3400.0 ± 495 |
| 0 | 3400.0 ± 495 |
| 1 | 3400.0 ± 495 |
| 2 | 3400.0 ± 495 |
| 3 | 3400.0 ± 495 |
| 4 | 3400.0 ± 495 |
| 5 | 3400.0 ± 495 |
| 6 | 3050.0 ± 0.0 |
| 7 | 3400.0 ± 495 |
| 8 | 3400.0 ± 495 |
| 9 | 3125.0 ± 884 |
| 10 | 3125.0 ± 884 |
| 11 | 3400.0 ± 495 |
| 12 | 3400.0 ± 495 |
| 13 | 3400.0 ± 495 |
| 14 | 3750.0 ± 0.0 |
| 15 | 3750.0 ± 0.0 |

Table 15. Mean IgG levels and standard deviations for the second trimester control heifers

| Week | Mean (± S.D.) |
|---|---|
| -2 | 2500.0 ± 0.0 |
| -1 | 2500.0 ± 0.0 |
| 0 | 2500.0 ± 0.0 |
| 1 | 2500.0 ± 0.0 |
| 2 | 2500.0 ± 0.0 |
| 3 | 2500.0 ± 0.0 |
| 4 | 2500.0 ± 0.0 |
| 5 | 2775.0 ± 389 |
| 6 | 2500.0 ± 0.0 |
| 7 | 2500.0 ± 0.0 |
| 8 | 2500.0 ± 0.0 |
| 9 | 2775.0 ± 389 |
| 10 | 2775.0 ± 389 |
| 11 | 2775.0 ± 389 |
| 12 | 2775.0 ± 389 |
| 13 | 2775.0 ± 389 |
| 14 | 2775.0 ± 389 |
| 15 | 2775.0 ± 389 |

Table 16: Mean IgG levels and standard deviations for the third trimester control heifers

| Week | Mean (± S.D.) |
|---|---|
| -2 | 3750.0 ± 0.0 |
| -1 | 3750.0 ± 0.0 |
| 0 | 3750.0 ± 0.0 |
| 1 | 3750.0 ± 0.0 |
| 2 | 3400.0 ± 495.0 |
| 3 | 3400.0 ± 495 |
| 4 | 3750.0 ± 0.0 |
| 5 | 3750.0 ± 0.0 |
| 6 | 3400.0 ± 495 |
| 7 | 3400.0 ± 495 |
| 8 | 3050.0 ± 0.0 |
| 9 | 3050.0 ± 0.0 |
| 10 | 3050.0 ± 0.0 |
| 11 | 3400.0 ± 495 |
| 12 | 3050.0 ± 0.0 |
| 13 | 3050.0 ± 0.0 |
| 14 | 3050.0 ± 0.0 |
| 15 | 3050.0 ± 0.0 |

Table 17: Analysis of variance table for IgI

| Stratum | DF | SS | SS% | MS | VR |
|---|---|---|---|---|---|
| Between Groups | 3 | 2.55 | 0.34 | 0.85 | 5.34 |
| Periods | 17 | 169.49 | 22.49 | 9.97 | 62.64 |
| Between Animals | 5 | 401.57 | 53.64 | 80.31 | 504.57 |
| Group/Period Interaction | 51 | 11.58 | 1.55 | 0.23 | 1.43 |
| Group/Animal Interaction | 15 | 17.09 | 2.28 | 1.14 | 7.16 |
| Period/Animal Interaction | 85 | 107.24 | 14.33 | 1.26 | 7.93 |
| Residual | 249 | 39.79 | 5.32 | 0.16 | |

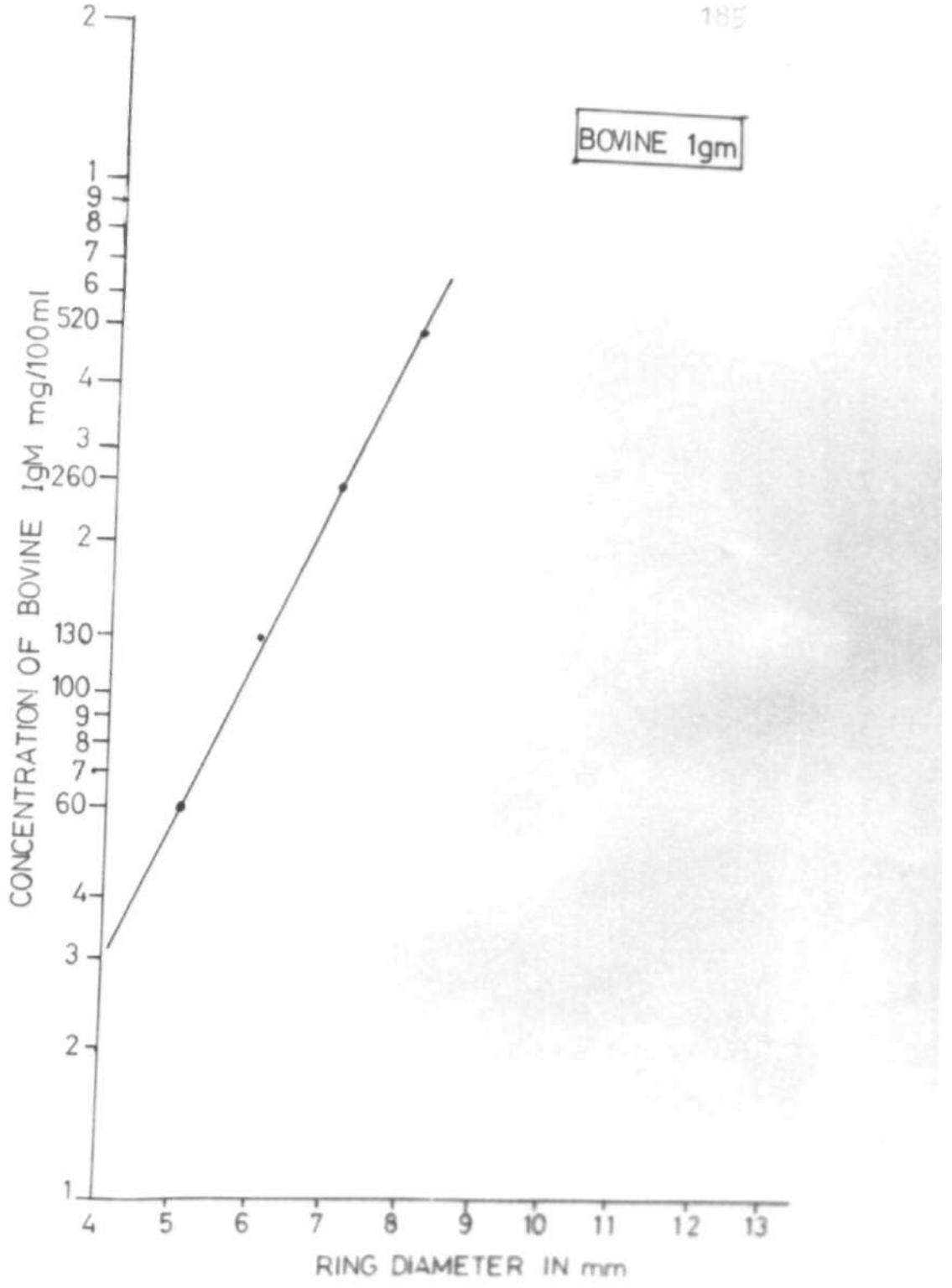
Table 18: Analysis of variance table for IgG

| Stratum | DF | SS | SSX | MS | VR |
|---|---|---|---|---|---|
| Between Groups | 3 | 16.93 | 5.94 | 5.64 | 55.18 |
| Periods | 17 | 60.14 | 31.10 | 3.54 | 34.61 |
| Between Animals | 5 | 103.94 | 36.47 | 20.79 | 203.35 |
| Group/Period Interaction | 51 | 13.16 | 4.62 | 0.26 | 2.53 |
| Group/Animal Interaction | 15 | 17.39 | 6.10 | 1.16 | 11.34 |
| Period/Animal Interaction | 85 | 48.55 | 17.03 | 0.57 | 5.54 |
| Residual | 249 | 25.45 | 8.93 | 0.10 | |

184

Figure 1

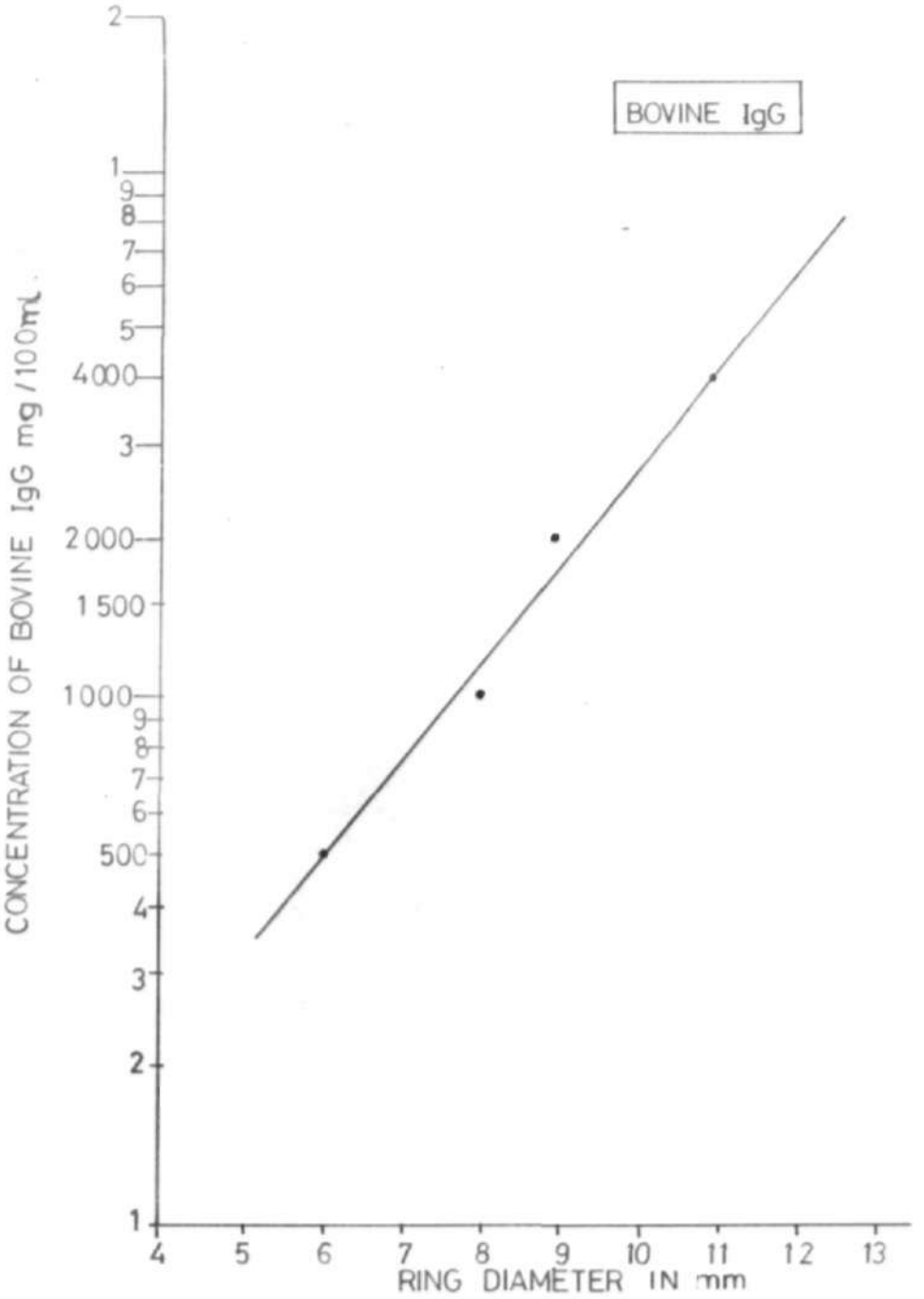
Standard IgM curve


BOVINE 1gm
CONCENTRATION OF BOVINE IgM mg/100ml
2
1
9
8
7
6
520
4
3
260
2
130
100
9
8
7
60
4
3
2
1
4
5
6
7
8
9
10
11
12
13
RING DIAMETER IN mm

186

Figure 2

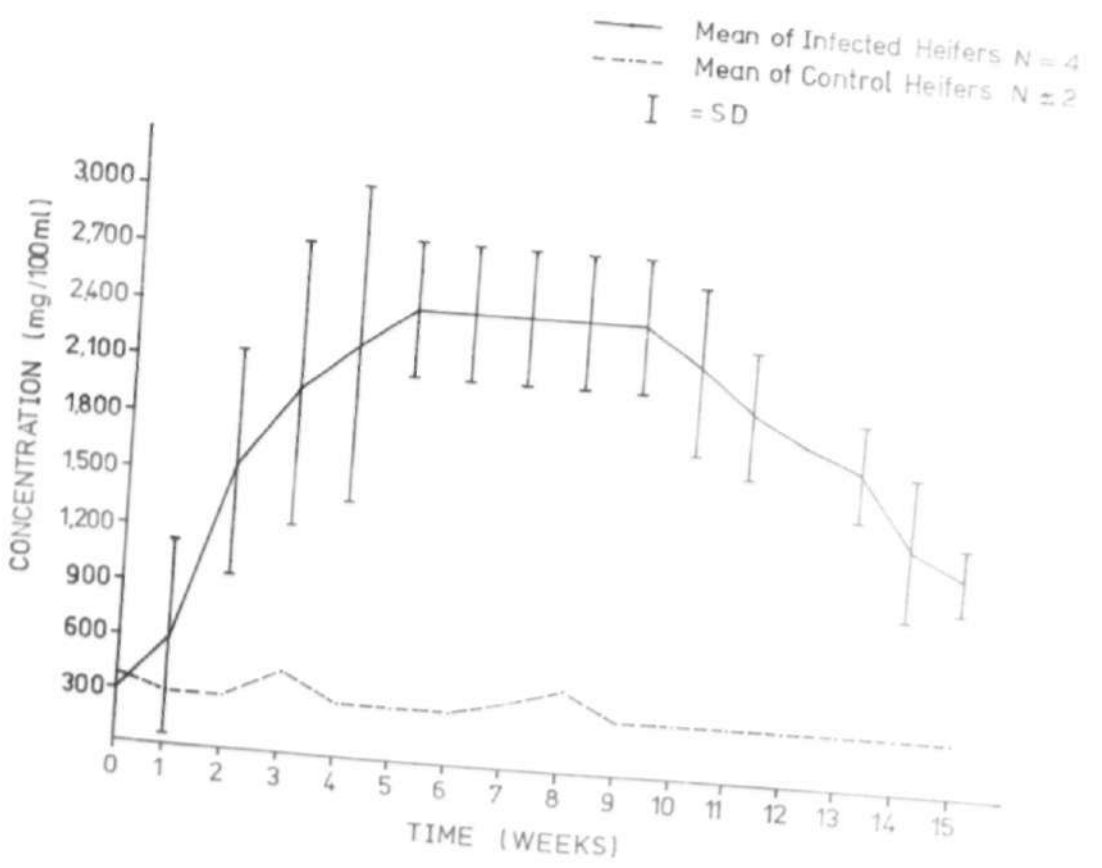
Standard IgG curve


BOVINE IgG
CONCENTRATION OF BOVINE IgG mg/100mL
2
1
9
8
7
6
5
4000
3
2000
1500
1000
9
8
7
6
500
4
3
2
1
4
5
6
7
8
9
10
11
12
13
RING DIAMETER IN mm

188

Figure 3

Mean IgM levels in non-pregnant heifers infected with T. vivax close to the time of estrus and that of the non-infected controls

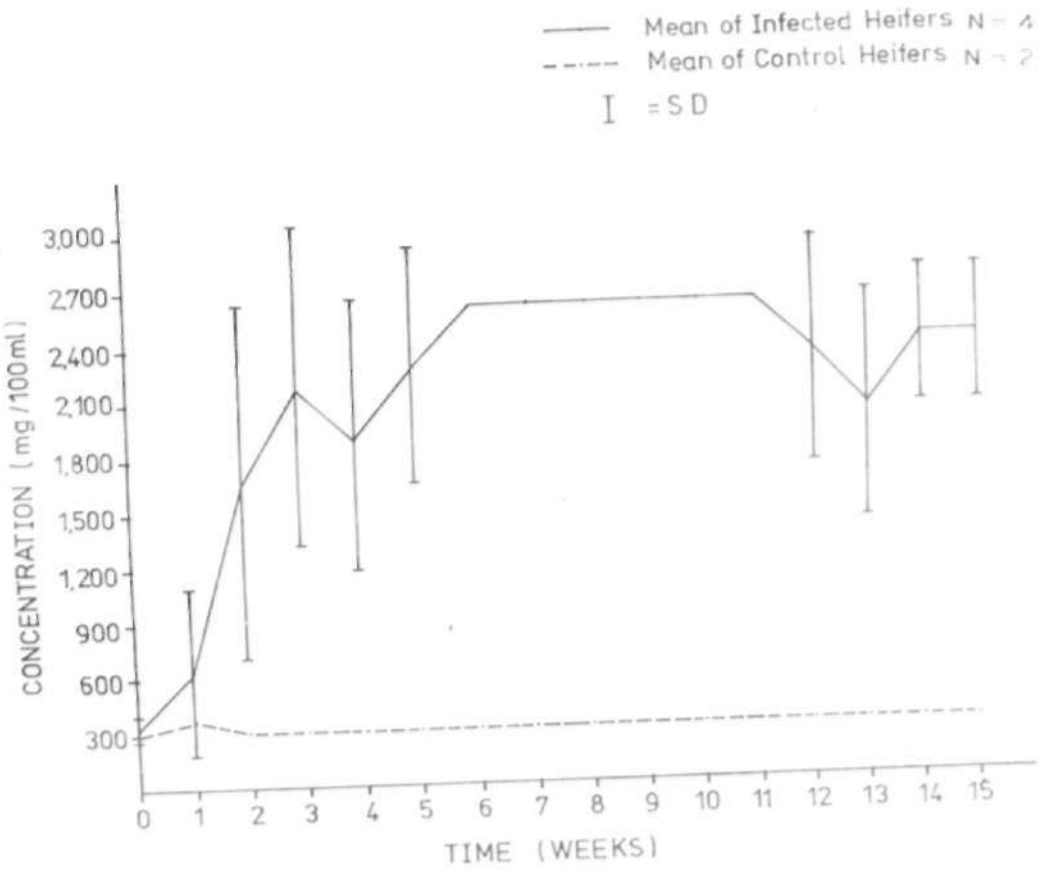

Mean of Infected Heifers N = 4
Mean of Control Heifers N = 2
I = SD
CONCENTRATION (mg/100ml)
3,000
2,700
2,400
2,100
1,800
1,500
1,200
900
600
300
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
TIME (WEEKS)

190

190

Figure 4

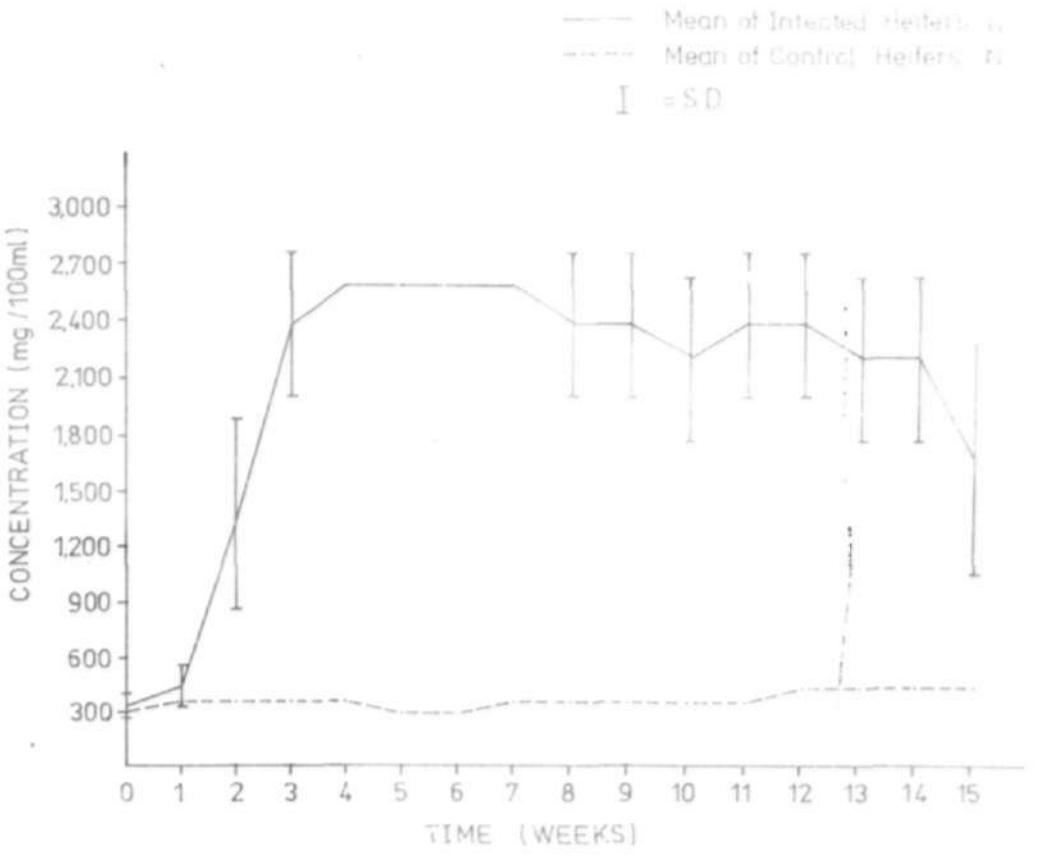
Mean Ig. levels in pregnant heifers infected during the first trimester with T. vivax and that of the non-infected pregnant controls


—— Mean of Infected Heifers N = 4
– – – – Mean of Control Heifers N = 2
I = S D
CONCENTRATION ( mg / 100ml )
3,000
2,700
2,400
2,100
1,800
1,500
1,200
900
600
300
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
TIME (WEEKS)

192

Figure 5

Mean Ig. levels in pregnant heifers infected during the second trimester with T. vivax and that of the non-infected pregnant controls


Mean of Infected Heifers (n
Mean of Control Heifers (n
I = S.D.
CONCENTRATION (mg / 100ml)
3,000
2,700
2,400
2,100
1,800
1,500
1,200
900
600
300
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
TIME (WEEKS)

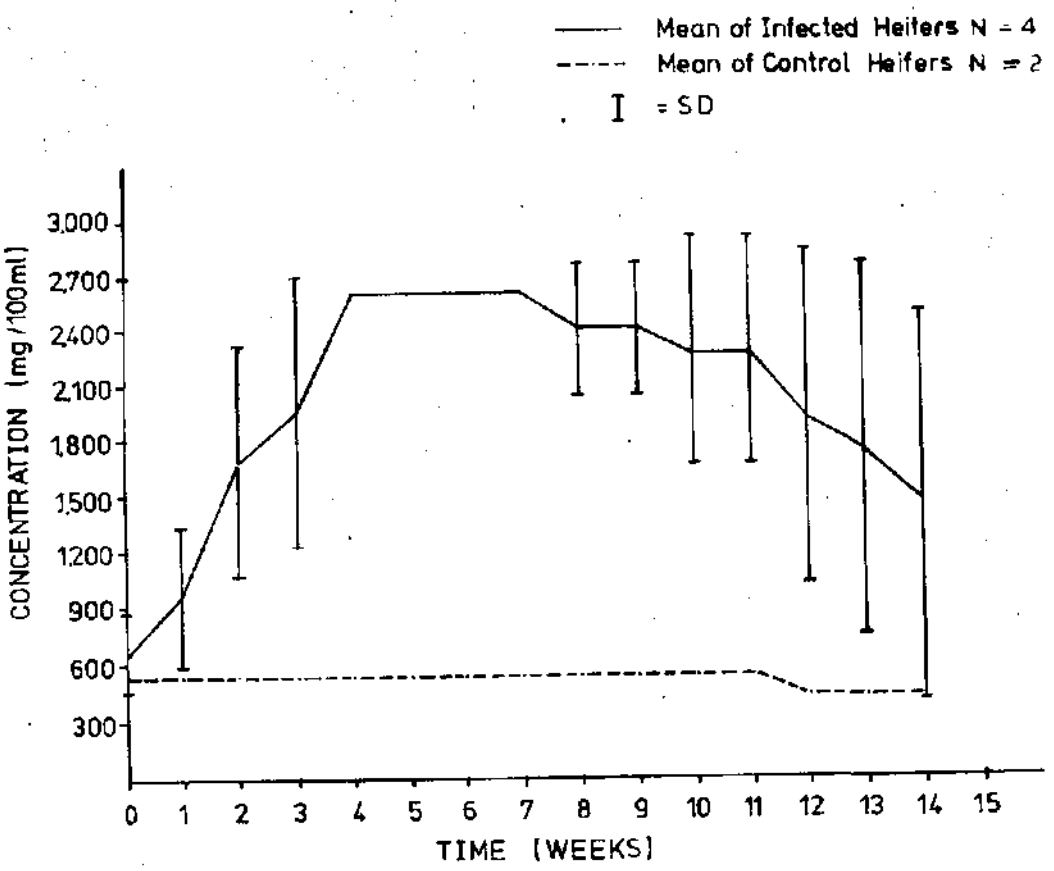
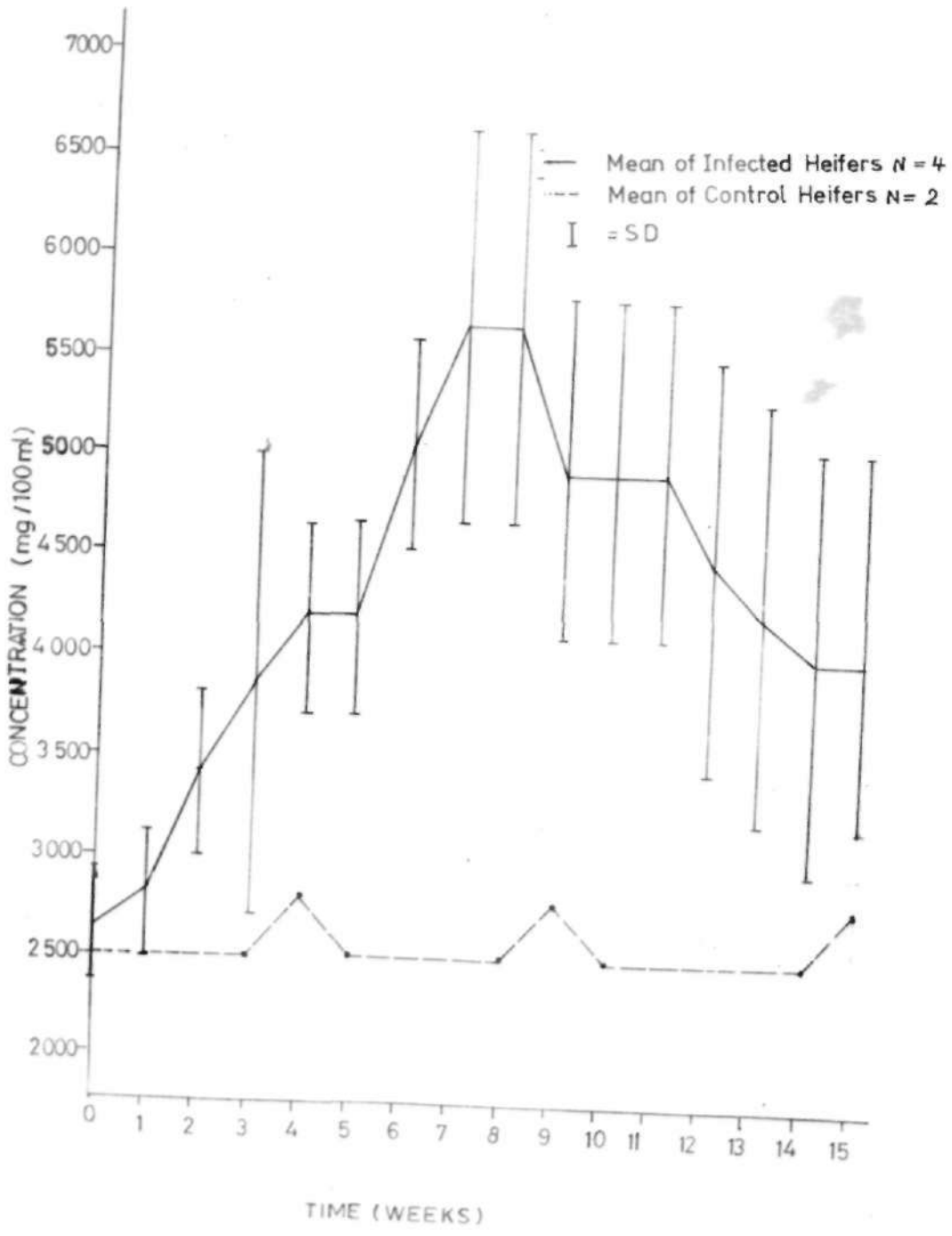

Mean of Infected Heifers N = 4
Mean of Control Heifers N = 2
I = SD
CONCENTRATION (mg/100ml)
3,000
2,700
2,400
2,100
1,800
1,500
1,200
900
600
300
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
TIME (WEEKS)

Figure 7

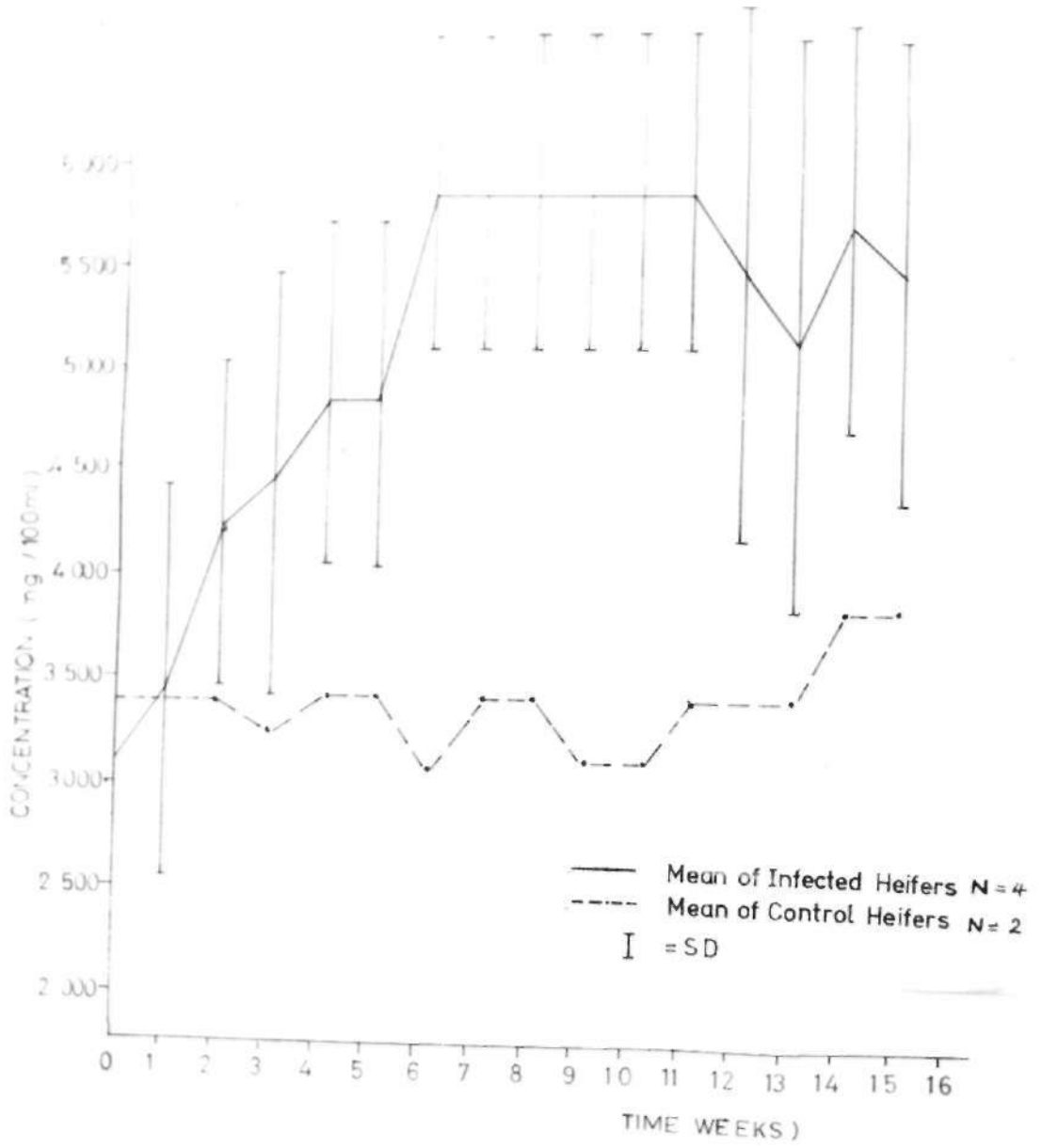
Mean IgG levels in non-pregnant heifers infected with T. vivax close to the time of estrus and that of the non-infected controls


Mean of Infected Heifers N = 4
Mean of Control Heifers N= 2
I = S D
CONCENTRATION (mg / 100 ml)
7000
6500
6000
5500
5000
4 500
4 000
3 500
3000
2500
2000
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
TIME (WEEKS)

198

Figure 8

Mean IgG levels in pregnant heifers infected during the first trimester with T. vivax and that of the non-infected pregnant controls

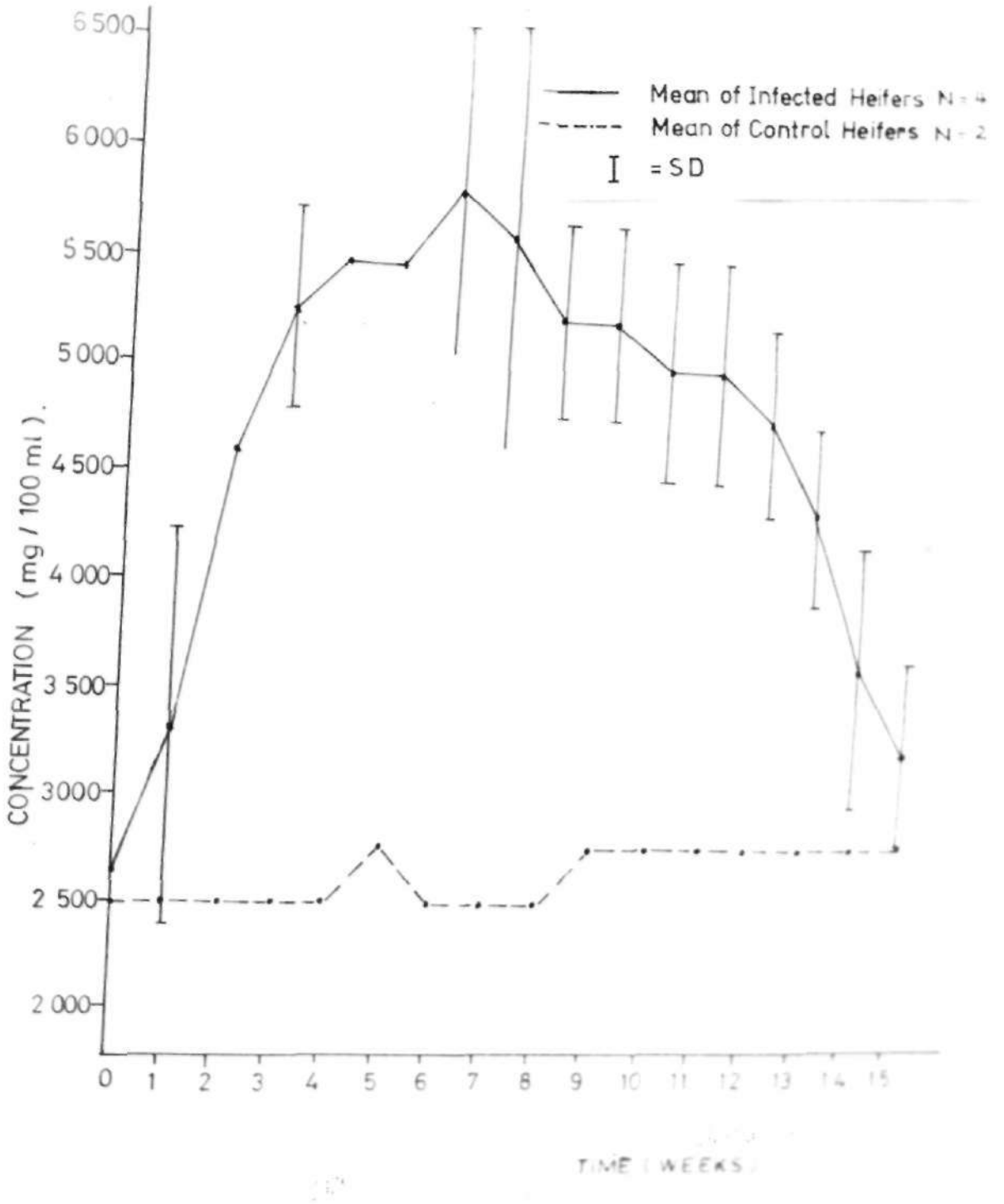

5 000
4 500
4 000
3 500
3 000
2 500
2 000
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16
TIME WEEKS)
Mean of Infected Heifers N = 4
Mean of Control Heifers N = 2
I = SD

200

200

Figure 9

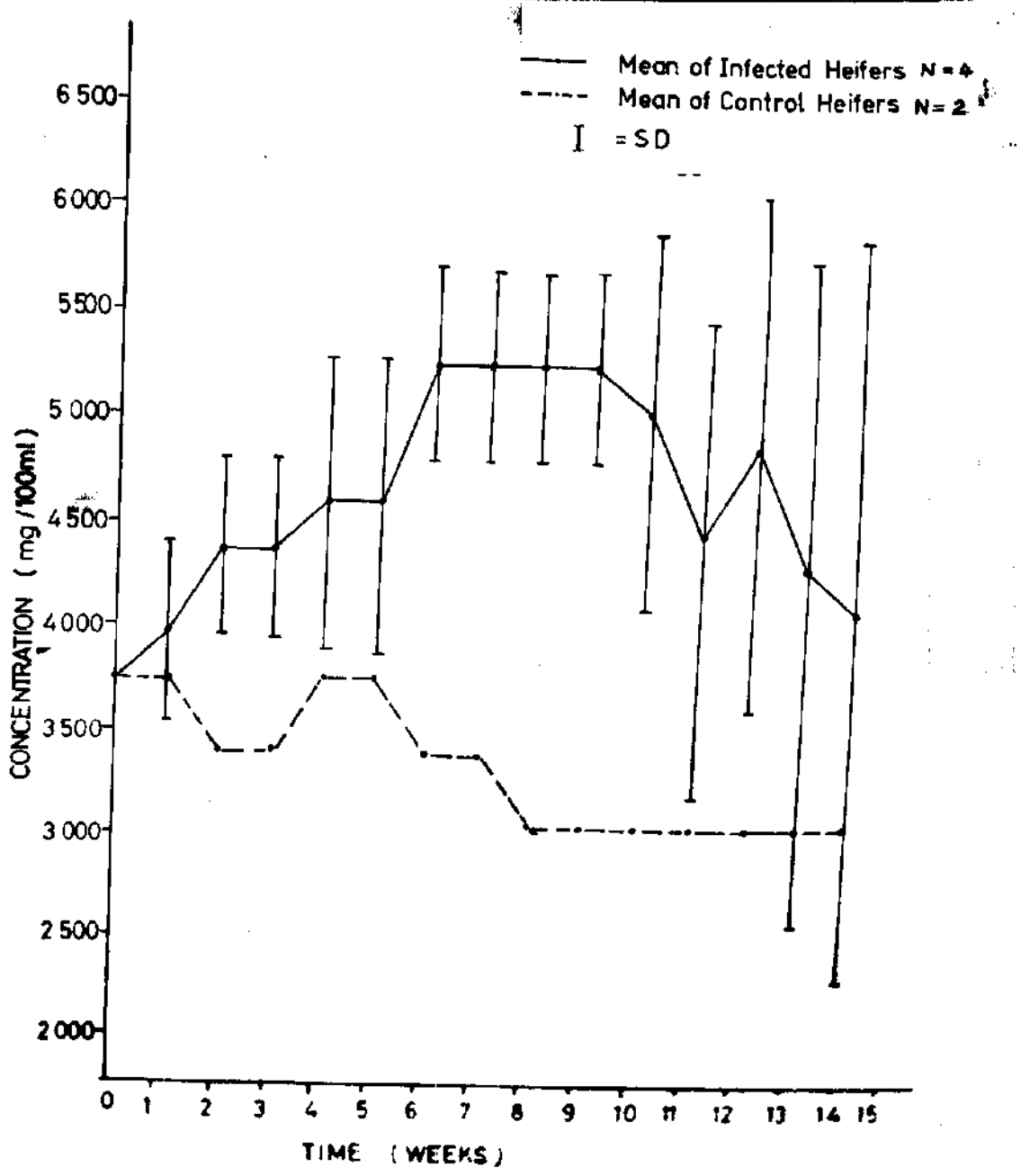
Mean IgG levels in pregnant heifers infected during the second trimester with T. vivax and that of the non-infected pregnant controls


Mean of Infected Heifers N = 4
Mean of Control Heifers N = 2
I = SD
CONCENTRATION (mg / 100 ml)
6 500
6 000
5 500
5 000
4 500
4 000
3 500
3000
2 500
2 000
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
TIME ( WEEKS )

202

Figure 10

Mean IgG levels in pregnant heifers infected during the third trimester with T. vivax and that of the non-infected pregnant controls


Mean of Infected Heifers N = 4
Mean of Control Heifers N = 2
I = S D
6 500
6 000
5 500
5 000
4 500
4 000
3 500
3 000
2 500
2 000
CONCENTRATION ( mg / 100ml )
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15
TIME ( WEEKS )

PART V

# ROLE OF T. VIVAX INFECTION OF FEMALE REPRODUCTION IN CATTLE:

*General discussion*

Clinical manifestations of trypanosomiasis in cattle produced by pathogenic trypanosomiasis are similar (Saror, 1975). Clinical signs include intermittent pyrexia, weight loss, debilitation, weakness and anemia. The course of the trypanosomiasis in this work varied in the individual heifers and dependent on the reproductive status of the animal.

Results of this study show that the Zebu cattle can withstand a single T. vivax infection. A mild course of infection was followed generally. This was probably due to the good nutrition and management the heifers were under. So, if good nutrition and proper management practice is followed, the widely held view that bovine trypanosomiasis is one of the principal factors restricting development of livestock industry in Nigeria (Leefland, 1974) may be disproved. The problem however is that concurrent infections such as anaplasmosis, babesiosis, heartwater and helminthiasis tend to complicate the picture. In this work, these concurrent infections were properly taken care of by regular dippings and prophylactic helminth control. Thus the effects of the trypanosome alone were more clearly observed in the animals and the much publicised weight loss, debility, weakness, anemia and eventual death were, to a very large extent, reduced. It is of interest to note that of the 16 heifers infected, only one died of the infection.

The extent of clinical manifestation of the infection varied generally more with the reproductive status of the heifer.

Of the 4 treatment groups, the non-pregnant heifers and heifers in the third trimester pregnancy showed more severe debilitation, anemia, weakness and lethargy. The observation was rather surprising especially in those non-pregnant heifers. While it may be easy to explain these observations in heifers infected during the third trimester those of non-pregnant heifers are difficult to explain. During the third trimester the heifers were stressed by the advanced pregnancy and T. vivax infection tended to add to this stress and therefore the clinical manifestations of the disease were more pronounced. It is not understood why non-pregnant heifers showed more severe signs of the disease than heifers in the first and second trimester pregnancy. However, it may not be unconnected with the hormonal changes associated with pregnancy. These changes, though not understood, may have improved the defence mechanisms of the heifers to T. vivax infection.

The finding of differences in the anemia produced by the infection is significant. Several workers (Fiennes, 1954; Naylor, 1971; Losos et al., 1973; Murray et al., 1974; Saror, 1975; Esievo, 1980) have

demonstrated anemia due to bovine trypanosomiasis. Some of these workers have been able to postulate the mechanisms involved in the production of the anemia. These include the hemodilution theory of Naylor (1971), direct toxic action of trypanosomes on blood (Hawkins et al., 1975); accelerated erthrocytic destruction (Fiennes, 1954, Losos et al., 1973); immunologically mediated erythrocyte destruction (Fiennes, 1954, Mackenzie and Cruickshank, 1973; Esuruoso, (1974c) and production of neuraminidase which cleaves off surface sialic acid, thus rendering erythrocytes more prone to phagocytosis by the reticuloendothelial system (Esievo, 1980). Whatever the mechanism is involved, this present work has demonstrated clearly that it is affected by the reproductive status of the heifer as non-pregnant heifers have more pronounced anemia when compared to pregnant heifers experimentally infected during the first and second trimester. The fall in PCV in heifers near term in both infected and non-infected may be due to the rather large amounts of blood being shunted to the placenta and hemodilution as the elevated estrongen levels at this time leads to water retention (Turner and Bagnara, 1976). Similar observation has been made by Hewett (1974).

The parasitemia in all the treatment groups was similar. The initial parasitemia was very high. Subsequently it showed fluctuations but lower than the initial. This reduced level of parasitemia may be a measure of the combating ability of the animals. However, the subsequent waves of parasitemia were higher in the individual animals in the non-pregnant group and heifers in the third trimester pregnancy than those in the first and second trimesters. In most of the infected heifers, it was observed that self cure occurred in the heifers after the first 3 months. In some animals, it was also observed that after abortion or premature delivery, the animals were better able to handle the infection as the parasitemia tended to drop or disappeared.

On the immunoglobulin M and G levels, it is clear from the results that there were significant increases over the preinfection level in all the groups of animals. The test was only a quantitative test, it is not known for sure how much of the immunoglobulins were directed against the trypanosome antigens. However, work by other authors (Seed et al., 1969) has shown that some of this tremendous production of IgM seems to be directed specifically against the trypanosomes while others may be heterophils (Houba et al., 1969; Mackenzie et al., 1973) or directed against autoantigens

(Mansfield and Kreir, 1972; Mackenzie et al., 1973). The persistent high levels of IgM may have resulted from continuous antigenic stimulation. Another factor that may be responsible for the high levels of IgM is the rather continuous antigenic variation characteristics of Trypanosome spp. Each new antigenic variant elicits new immunologic response culminating in the persistently high levels of IgM. The present work has shown that the immunologic response to T. vivax infection is dependent on reproductive status of the heifers as the response of the infected non-pregnant heifers and heifers infected during the third trimester were inferior to those of heifers infected during the first and second trimester.

The effects of T. vivax infection on reproduction in heifers have been discussed in the various chapters. In general the effects include conception failure, anestrus, abortion in the first trimester, premature and weak calving in the third trimester. Since efficient reproduction is a very important part of cattle production, it is imperative that the role of trypanosome infection in cattle should be viewed with all seriousness.

Conception failure and infertility in cattle population in this country have not been thoroughly studied. No one knows for sure the magnitude of the problems, nor all the cause(s). Trichomonas foetus, known to cause conception failure in some parts of the world has not been positively identified as a cause in our indigenous cattle. It is therefore important that the finding of conception failure and anestrus resulting from T. vivax infection be taken seriously and adequate steps taken to recommend prophylactic course of action especially in the endemic areas. It may be stated here that the economic significance of this finding is high. The picture is complicated by the fact that these conception failures are again followed by a long period of anestrous in the infected heifers. Thus one can see why calving intervals ranging from 16 months (Fricke, 1964) - 24 months (Nuru, 1974) have been quoted

for our indigenous cattle population in the northern states of Nigeria while a calving interval as high as 27 months has been reported in nomadic herds (Pullan, 1979).

The question is why do cows in endemic areas become pregnant since they are in constant tse-tse fly challenge. The possible answer may be that cattle in endemic areas are challenged early in life. As a result, some die while others survive. The animals that survive acquire some immunity to the trypanosome infection with mild subsequent infections. However, since the majority of our cattle population are in the traditional breeding zones of the Sahel and Sudan vegetation, it is only those that migrate to infested areas that are affected.

It is true that only one of the 4 infected heifers in the first trimester aborted. Similar early abortions have been observed by Murray et al., (1981) in Zebu cattle exposed to natural field challenge.

The premature deliveries seen in the third trimester heifers in this work is a new dimension in our ever growing knowledge of bovine trypanosomiasis. The fact that this had not been previously reported, may have been due to the fact that heavily pregnant cows are not usually used for experiments.

The hypotheses to explain the role T. vivax infection on reproduction in heifers are illustrated in figure 1 and 2. From these it can be seen that in non-pregnant heifers clinical trypanosomiasis results in the disruption of reproductive process. It is postulated that the trypanosomes or their products (see part II) cause conception failure by affecting the conceptus directly or by indirectly affecting the uterine environment by elevating the body temperature thus making it impossible for implanatation to occur or causing death of the conceptus. The observed anestrus is postulated to be caused by the effect of the infection on the pituitary gland.

In pregnant heifers (figure 2) infection during the first trimester results in sporadic abortions. These abortions are postulated to have resulted from fetal infection and subsequent death. However in some heifers, abortion do not occur. Here it is postulated that the organisms still cross the placental barriers leading to fetal infection. For some unknown reasons the fetuses get rid of the infection and the pregnancy progresses rather normally leading to the delivery of viable but weak calves.

In heifers infected during the second trimester, it is postulated that the trypanosomes may have still crossed the placental barriers. However, the fetuses

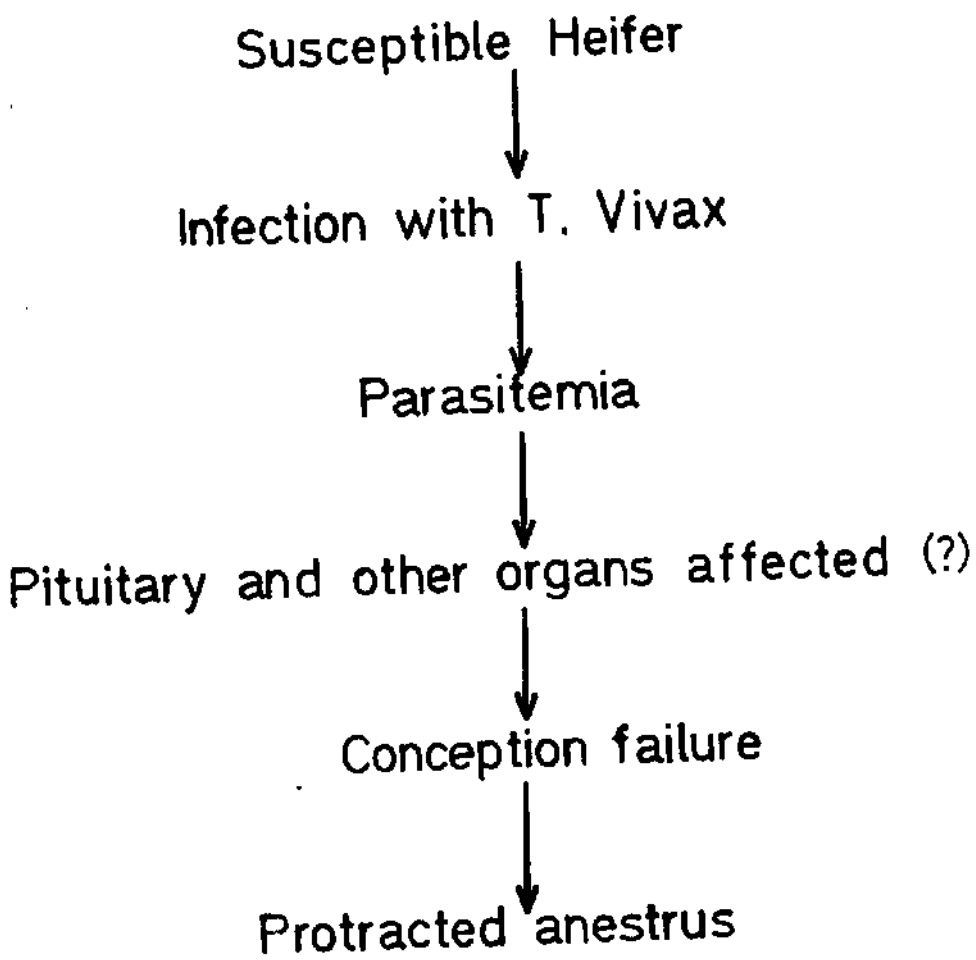
at this time do not develop parasitemia and pregnancy is not affected. In this group viable calves are born.

In the third trimester heifers, infection with T. vivax results in fetal parasitemia leading to premature deliveries, stillbirths and deliveries of non-viable calves. In these calves parasitemia is demonstrable is some while in others it is not.

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Figure 1

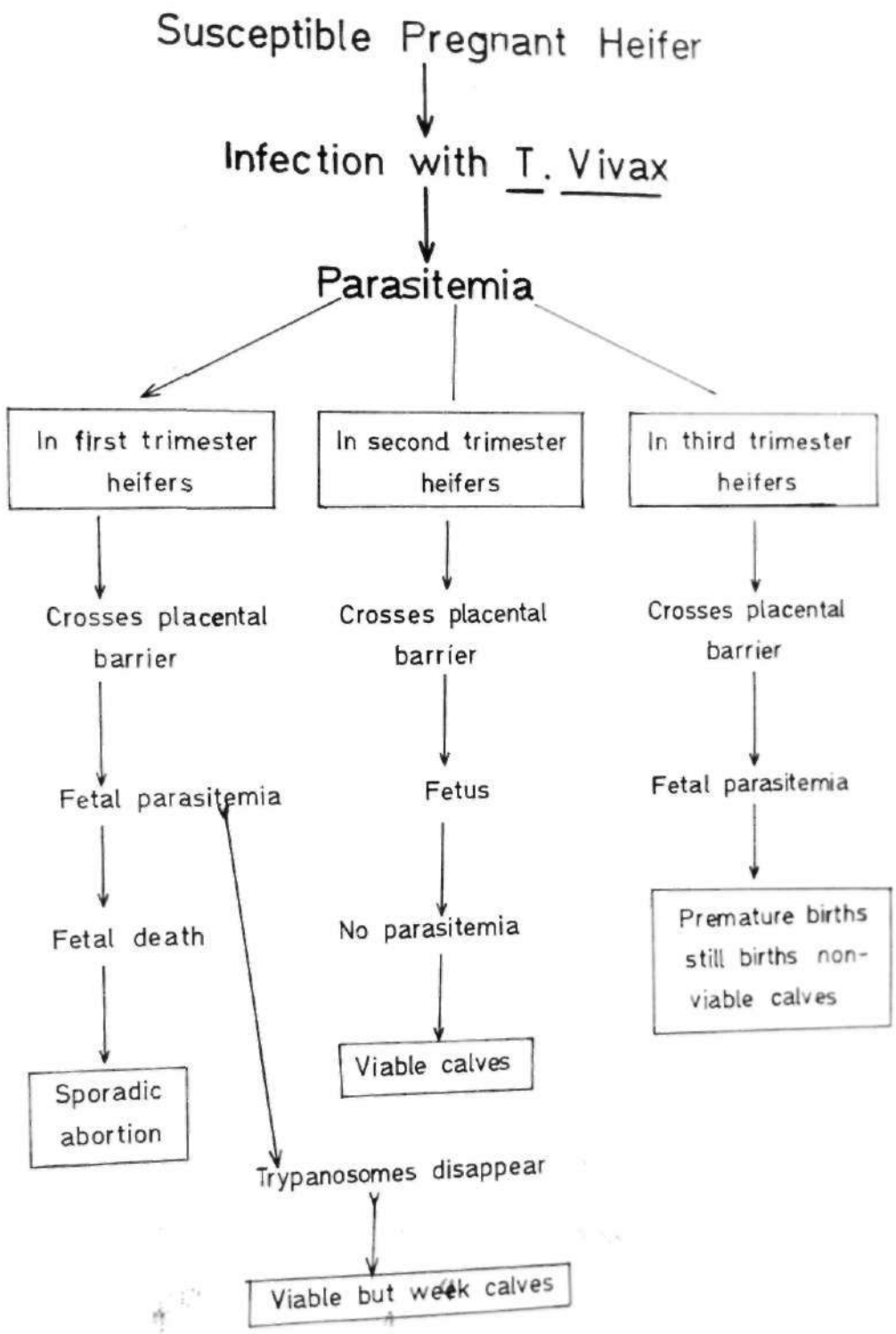
Hypothesis to explain the effects of T. vivax infection on reproduction in heifers.


Susceptible Heifer
Infection with T. Vivax
Parasitemia
Pituitary and other organs affected (?)
Conception failure
Protracted anestrus

216

Figure 2

Hypothesis to explain the effects of
T. vivax infection on pregnancy in heifers


Susceptible Pregnant Heifer
Infection with T. Vivax
Parasitemia
In first trimester heifers
In second trimester heifers
In third trimester heifers
Crosses placental barrier
Crosses placental barrier
Crosses placental barrier
Fetal parasitemia
Fetus
Fetal parasitemia
Fetal death
No parasitemia
Premature births still births non-viable calves
Sporadic abortion
Viable calves
Trypanosomes disappear
Viable but week calves

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APPENDIX 1

CHANGES IN RECTAL TEMPERATURE, PCV AND PARASITEMIA IN NONPREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH TRYPANOSOMA VIVAX CLOSE TO THE TIME OF ESTRUS

HEIFER NO. L 31

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 44.0% | NPF | T. mutans few |
| - 29 | 38.5°C | 44.0% | NPF | T. mutans few |
| - 22 | 38.6°C | 42.5% | NPF | T. mutans few |
| - 15 | 38.5°C | 42.5% | NPF | NPF |
| - 8 | 38.5°C | 42.5% | NPF | NPF |
| - 3 | 38.5°C | 43.0% | NPF | NPF |
| 0 | 38.5°C | 43.0% | NPF | NPF T. Vivax T |
| 1 | 38.5°C | 42.5% | NPF | NPF |
| 2 | 38.5°C | 4 .5% | NPF | NPF |
| 3 | 38.6°C | 42.5% | NPF | NPF |
| 4 | 39.5°C | 42.0% | Tryps + | NPF |
| 5 | 39.6°C | 42.0% | Tryps + | T. vivax |
| 6 | 39.7°C | 42.0% | Tryps + | T. vivax Breed |
| 7 | 39.8°C | 42.5% | Tryps +++ | T. vivax |
| 8 | 39.8°C | 41.0% | Tryps ++++ | T. vivax |
| 9 | 39.8°C | 41.5% | Tryps ++++ | T. vivax |
| 10 | 40.0°C | 40.0% | Tryps +++ | T. vivax |
| 11 | 40.0°C | 39.0% | Tryps ++++ | T. vivax |
| 12 | 40.5°C | 39.0% | Tryps ++++ | T. vivax |
| 13 | 40.5°C | 38.5% | Tryps ++++ | T. vivax |
| 14 | 40.5°C | 38.5% | Tryps +++ | T. vivax |
| 15 | 40.0°C | 38.0% | Tryps ++ | T. vivax |
| 16 | 40.5°C | 36.0% | Tryps ++ | T. vivax |
| 17 | 40.0°C | 36.0% | Tryps + | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.8°C | 35.0% | Tryps + | T. vivax |
| 21 | 39.5°C | 30.0% | Tryps + | T. vivax |
| 25 | 39.5°C | 28.5% | Tryps ++ | T. vivax |
| 28 | 40.0°C | 26.0% | Tryps ++ | T. vivax |
| 31 | 40.0°C | 26.0% | Tryps + | T. vivax |
| 36 | 39.9°C | 25.0% | Tryps + | T. vivax |
| 39 | 38.8°C | 25.0% | NPF | NPF |
| 42 | 38.6°C | 18.0% | Tryps ++ | T. vivax |
| 44 | 38.4°C | 18.5% | Tryps + | T. vivax |
| 49 | 39.8°C | 15.0% | Tryps + | NPF |
| 51 | 39.9°C | 17.5% | Tryps + | T. vivax |
| 53 | 39.8°C | 18.0% | Tryps + | NPF |
| 58 | 40.0°C | 18.0% | Tryps + | NPF |
| 60 | 38.9°C | 19.0% | NPF | NPF |
| 63 | 38.4°C | 20.5% | NPF | NPF |
| 65 | 39.0°C | 20.0% | NPF | NPF |
| 70 | 39.9°C | 21.0% | Tryps ++ | T. vivax |
| 72 | 39.8°C | 20.5% | NPF | NPF |
| 77 | 39.6°C | 22.0% | NPF | NPF |
| 79 | 39.4°C | 25.0% | NPF | NPF |
| 81 | 38.8°C | 25.5% | NPF | NPF |
| 85 | 38.9°C | 26.0% | NPF | NPF |
| 86 | 38.8°C | 26.0% | Tryps + | NPF |
| 88 | 38.8°C | 26.0% | NPF | NPF |
| 91 | 40.0°C | 26.5% | NPF | NPF |
| 93 | 39.8°C | 27.0% | Tryps + | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 95 | 38.6°C | 21.0% | Tryps + | T. vivax |
| 98 | 39.0°C | 21.5% | NPF | NPF |
| 100 | 38.8°C | 22.5% | NPF | NPF |
| 102 | 38.8°C | 24.0% | NPF | NPF |
| 109 | 38.6°C | 25.0% | NPF | NPF |
| 115 | 38.5°C | 25.0% | NPF | NPF |
| 122 | 38.5°C | 25.5% | NPF | NPF |
| 129 | 38.5°C | 26.0% | NPF | NPF |
| 135 | 38.5°C | 26.0% | NPF | NPF |
| 157 | 38.4°C | 26.0% | NPF | NPF |
| 164 | 38.4°C | 26.5% | NPF | NPF |
| 171 | 38.5°C | 27.0% | NPF | NPF |

HEIFER NO. L 25

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 45.0% | NPF | T. mutans few |
| - 29 | 38.4°C | 45.0% | NPF | T. mutans few |
| - 22 | 38.4°C | 46.0% | NPF | T. mutans few |
| - 15 | 38.5°C | 45.5% | NPF | NPF |
| - 8 | 38.5°C | 45.0% | NPF | NPF |
| - 3 | 38.5°C | 45.0% | NPF | NPF |
| 0 | 38.5°C | 45.0% | NPF | NPF T. vivax |
| 1 | 38.5°C | 45.0% | NPF | NPF |
| 2 | 38.5°C | 45.0% | NPF | NPF |
| 3 | 38.7°C | 44.5% | NPF | NPF |
| 4 | 38.8°C | 45.0% | NPF | NPF |
| 5 | 38.8°C | 45.0% | Tryps + | NPF Breed |
| 6 | 38.8°C | 44.0% | Tryps ++ | T vivax |
| 7 | 39.0°C | 43.0% | Tryps ++ | T. vivax |
| 8 | 39.5°C | 42.0% | Tryps +++ | T. vivax |
| 9 | 39.9°C | 40.0% | Tryps ++++ | T. vivax |
| 10 | 39.8°C | 40.0% | Tryps ++++ | T. vivax |
| 11 | 39.9°C | 39.0% | Tryps ++++ | T. vivax |
| 12 | 40.0°C | 36.0% | Tryps +++ | T. vivax |
| 13 | 40.0°C | 36.5% | Tryps ++++ | T. vivax |
| 14 | 40.0°C | 35.0% | Tryps +++ | T. vivax |
| 15* | 40.5°C | 35.5% | Tryps ++ | T. vivax |
| 16 | 40.0°C | 31.0% | Tryps ++ | T. vivax |
| 17 | 40.0°C | 31.0% | Tryps ++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.8°C | 30.0% | Tryps ++ | T. vivax |
| 21 | 39.9°C | 28.0% | Tryps +++ | T. vivax |
| 25 | 39.9°C | 22.0% | Tryps ++ | T. vivax |
| 28 | 39.8°C | 22.0% | Tryps + | T. vivax |
| 31 | 39.6°C | 21.0% | Tryps + | NPF |
| 36 | 38.8°C | 20.0% | NPF | NPF |
| 39 | 38.9°C | 18.0% | Tryps ++ | T. vivax |
| 42 | 38.9°C | 18.5% | Tryps + | T. vivax |
| 44 | 39.0°C | 16.0% | Tryps + | T. vivax |
| 49 | 40.5°C | 14.0% | Tryps ++ | T. vivax |
| 51 | 38.5°C | 13.5% | Tryps + | T. vivax |
| 53 | 38.3°C | 13.0% | Tryps + | T. vivax |
| 58 | 38.6°C | 16.0% | NPF | NPF |
| 60 | 38.5°C | 18.5% | NPF | NPF |
| 63 | 38.4°C | 19.0% | Tryps + | NPF |
| 65 | 39.0°C | 22.0% | NPF | NPF |
| 70 | 38.8°C | 22.5% | NPF | NPF |
| 72 | 38.5°C | 25.0% | NPF | NPF |
| 77 | 38.6°C | 26.0% | NPF | NPF |
| 79 | 38.5°C | 26.0% | NPF | NPF |
| 81 | 38.5°C | 26.5% | NPF | T. mutans few |
| 85 | 38.4°C | 28.0% | NPF | T. mutans few |
| 85 | 38.5°C | 28.5% | NPF | T. mutans few |
| 88 | 38.5°C | 28.5% | NPF | NPF |
| 91 | 38.5°C | 28.5% | NPF | NPF |
| 93 | 38.6°C | 28.5% | NPF | NPF |

| Day | Temperature | PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 95 | 38.4°C | 29.0% | NPF | NPF |
| 98 | 38.4°C | 30.0% | NPF | NPF |
| 100 | 38.5°C | 30.0% | NPF | NPF |
| 102 | 38.4°C | 30.0% | NPF | NPF |
| 109 | 38.5°C | 30.0% | NPF | NPF |
| 115 | 38.5°C | 30.5% | NPF | NPF |
| 122 | 38.5°C | 30.5% | NPF | NPF |
| 129 | 38.5°C | 30.0% | NPF | NPF |
| 135 | 38.5°C | 30.0% | NPF | NPF |
| 157 | 38.6°C | 30 0% | NPF | NPF |
| 164 | 38.6°C | 31.0% | NPF | NPF |
| 171 | 38.4°C | 31.0% | NPF | NPF |

HEIFER NO. L 20

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.6°C | 42.5% | NPF | T. mutans few |
| - 29 | 38.4°C | 41.0% | NPF | T. mutans few |
| - 22 | 38.5°C | 40.5% | NPF | T. mutans few |
| - 15 | 38.4°C | 41.0% | NPF | NPF |
| - 8 | 38.4°C | 40.5% | NPF | NPF |
| - 3 | 38.5°C | 40.0% | NPF | NPF |
| 0 | 38.4°C | 40.0% | NPF | NPF T. vivax |
| 1 | 38.5°C | 40.0% | NPF | NPF |
| 2 | 38.5°C | 40.0% | NPF | NPF |
| 3 | 38.6°C | 40.5% | NPF | NPF |
| 4 | 38.8°C | 40.0% | Tryps + | NPF |
| 5 | 39.0°C | 39.0% | Tryps + | T. vivax |
| 6 | 39.0°C | 39.5% | Tryps + | T. vivax Breed |
| 7 | 39.0°C | 38.0% | Tryps ++ | T. vivax |
| 8 | 39.8°C | 38.0% | Tryps +++ | T. vivax |
| 9 | 39.9°C | 37.0% | Tryps ++++ | T. vivax |
| 10 | 39.9°C | 37.0% | Tryps ++++ | T. vivax |
| 11 | 40.0°C | 37.0% | Tryps ++++ | T. vivax |
| 12 | 40.5°C | 35.0% | Tryps ++++ | T. vivax |
| 13 | 40.0°C | 35.5% | Tryps +++ | T. vivax |
| 14 | 40.0°C | 35.5% | Tryps +++ | T. vivax |
| 15 | 39.9°C | 31.0% | Tryps +++ | T. vivax |
| 16 | 39.8°C | 30.0% | Tryps ++++ | T. vivax |
| 17 | 39.8°C | 30.0% | Tryps ++++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.8°C | 30.0% | Tryps +++ | T. vivax |
| 21 | 39.9°C | 30.0% | Tryps +++ | T. vivax |
| 25 | 39.0°C | 24.0% | Tryps ++ | T. vivax |
| 28 | 39.0°C | 24.0% | Tryps + | T. vivax |
| 31 | 39.5°C | 23.5% | Tryps + | T. vivax |
| 36 | 38.5°C | 19.5% | Tryps +++ | T. vivax |
| 39 | 38.9°C | 19.5% | Tryps +++ | T. vivax |
| 42 | 39.5°C | 18.0% | Tryps ++ | T. vivax |
| 44 | 39.0°C | 18.5% | Tryps ++ | T. vivax |
| 49 | 38.9°C | 19.0% | Tryps + | T. vivax |
| 51 | 39.0°C | 19.5% | Tryps + | T. vivax |
| 53 | 39.0°C | 19.5% | Tryps + | T. vivax |
| 58 | 38.8°C | 20.0% | Tryps + | NPF |
| 60 | 39.0°C | 20.0% | NPF | NPF |
| 63 | 39.4°C | 18.0% | NPF | NPF |
| 65 | 39.0°C | 18.0% | NPF | NPF |
| 70 | 38.5°C | 18.5% | NPF | NPF |
| 72 | 38.5°C | 19.0% | NPF | NPF |
| 77 | 39.9°C | 21.0% | Tryps + | NPF |
| 79 | 39.5°C | 21.5% | Tryps + | T. vivax |
| 81 | 39.0°C | 21.5% | NPF | NPF |
| 85 | 38.6°C | 22.0% | NPF | NPF |
| 86 | 38.6°C | 22.0% | NPF | NPF |
| 88 | 38.4°C | 22.0% | NPF | NPF |
| 91 | 38.5°C | 23.0% | NPF | NPF |
| 93 | 39.0°C | 22.5% | Tryps + | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 95 | 38.6°C | 25.0% | NPF | NPF |
| 98 | 38.5°C | 25.0% | NPF | NPF |
| 100 | 38.5°C | 25.5% | NPF | NPF |
| 102 | 38.4°C | 26.0% | NPF | NPF |
| 109 | 38.4°C | 25.5% | NPF | NPF |
| 115 | 38.4°C | 25.5% | NPF | NPF |
| 122 | 38.5°C | 26.0% | NPF | NPF |
| 129 | 38.6°C | 27.0% | NPF | NPF |
| 135 | 38.4°C | 26.5% | NPF | NPF |
| 157 | 38.5°C | 26.0% | NPF | NPF |
| 164 | 38.5°C | 26.0% | NPF | NPF |
| 171 | 38.4°C | 28.0% | NPF | NPF |

HEIFER NO. L.32

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.8°C | 41.0% | NPF | T. mutans few |
| - 29 | 38.5°C | 40.0% | NPF | T. mutans few |
| - 22 | 38.6°C | 40.0% | NPF | T. mutans few |
| - 15 | 38.4°C | 40.5% | NPF | T. mutans few |
| - 8 | 38.6°C | 40.5% | NPF | NPF |
| - 3 | 38.5°C | 40.0% | NPF | NPF |
| 0 | 38.4°C | 40.0% | NPF | NPF.T. vivax |
| 1 | 38.5°C | 40.5% | NPF | NPF |
| 2 | 38.4°C | 40.5% | NPF | NPF |
| 3 | 38.6°C | 40.0% | NPF | NPF |
| 4 | 38.8°C | 41.0% | NPF | NPF |
| 5 | 38.8°C | 40.0% | Tryps + | T. vivax |
| 6 | 38.9°C | 40.5% | Tryps ++ | T. vivax |
| 7 | 39.8°C | 39.5% | Tryps ++ | T. vivax |
| 8 | 39.9°C | 39.0% | Tryps ++++ | T. vivax Breed |
| 9 | 39.9°C | 38.0% | Tryps ++++ | T. vivax |
| 10 | 39.9°C | 38.0% | Tryps ++++ | T. vivax |
| 11 | 40.0°C | 36.0% | Tryps ++++ | T. vivax |
| 12 | 40.0°C | 35.5% | Tryps +++ | T. vivax |
| 13 | 40.0°C | 35.5% | Tryps +++ | T. vivax |
| 14 | 40.0°C | 33.0% | Tryps ++ | T. vivax |
| 15 | 39.8°C | 33.0% | Tryps + | T. vivax |
| 16 | 39.8°C | 30.0% | Tryps + | T. vivax |
| 17 | 39.8°C | 30.0% | Tryps +++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.8°C | 29.0% | Tryps +++ | T. vivax |
| 21 | 39.9°C | 27.0% | Tryps ++++ | T. vivax |
| 25 | 40.0°C | 23.0% | Tryps +++ | T. vivax |
| 28 | 40.2°C | 23.0% | Tryps ++ | T. vivax |
| 31 | 39.8°C | 20.0% | Tryps + | T. vivax |
| 36 | 39.0°C | 20.0% | Tryps + | T. vivax |
| 39 | 38.8°C | 19.0% | Tryps ++ | T. vivax |
| 42 | 38.9°C | 18.0% | Tryps + | T. vivax |
| 44 | 38.6°C | 18.0% | Tryps + | T. vivax |
| 49 | 38.5°C | 16.0% | Tryps + | NPF |
| 51 | 38.6°C | 18.0% | Tryps + | T. vivax |
| 53 | 38.6°C | 19.0% | Tryps + | T. vivax |
| 58 | 37.0°C | 20.0% | NPF | NPF |
| 60 | 38.9°C | 17.0% | Tryps + | T. vivax |
| 63 | 39.0°C | 18.0% | NPF | NPF |
| 65 | 39.5°C | 20.5% | NPF | NPF |
| 70 | 39.0°C | 21.0% | NPF | NPF |
| 72 | 39.0°C | 21.5% | NPF | NPF |
| 77 | 38.9°C | 21.0% | Tryps + | NPF |
| 79 | 38.8°C | 21.0% | NPF | NPF |
| 81 | 38.8°C | 21.5% | NPF | NPF |
| 85 | 39.0°C | 23.0% | NPF | NPF |
| 86 | 38.5°C | 24.0% | NPF | NPF |
| 88 | 38.5°C | 24.0% | NPF | NPF |
| 91 | 38.5°C | 24.5% | NPF | NPF |
| 93 | 38.4°C | 25.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 95 | 38.5°C | 25.0% | NPF | NPF |
| 98 | 38.6°C | 25.5% | NPF | NPF |
| 100 | 38.5°C | 26.0% | NPF | NPF |
| 102 | 38.5°C | 26.0% | NPF | NPF |
| 109 | 38.5°C | 26.5% | NPF | NPF |
| 115 | 38.4°C | 26.0% | NPF | NPF |
| 122 | 38.6°C | 26.0% | NPF | NPF |
| 129 | 38.4°C | 25.5% | NPF | NPF |
| 135 | 38.4°C | 25.0% | NPF | NPF |
| 157 | 38.5°C | 26.0% | NPF | NPF |
| 164 | 38.5°C | 26.0% | NPF | NPF |
| 171 | 38.5°C | 26.0% | NPF | NPF |

HEIFER NO. L 66 (CONTROL)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 42.0% | NPF | T. mutans few |
| - 29 | 38.4°C | 40.0% | NPF | T. mutans few |
| - 22 | 38.6°C | 40.5% | NPF | T. mutans few |
| - 15 | 38.4°C | 40.0% | NPF | T. mutans few |
| - 8 | 38.4°C | 40.0% | NPF | NPF |
| - 3 | 38.3°C | 41.0% | NPF | NPF |
| 0 | 38.5°C | 42.0% | NPF | NPF |
| 1 | 38.4°C | 41.5% | NPF | NPF |
| 2 | 38.5°C | 40.0% | NPF | NPF |
| 3 | 38.5°C | 40.5% | NPF | NPF |
| 4 | 38.5°C | 40.5% | NPF | NPF |
| 5 | 38.5°C | 41.5% | NPF | NPF |
| 6 | 38.4°C | 41.0% | NPF | NPF |
| 7 | 38.3°C | 40.0% | NPF | NPF |
| 8 | 38.6°C | 40.0% | NPF | NPF |
| 9 | 38.4°C | 40.0% | NPF | NPF |
| 10 | 38.5°C | 41.5% | NPF | NPF |
| 11 | 38.5°C | 41.0% | NPF | NPF |
| 12 | 38.5°C | 40.0% | NPF | NPF |
| 13 | 38.5°C | 40.0% | NPF | NPF |
| 14 | 38.5°C | 40.0% | NPF | NPF |
| 15 | 38.6°C | 40.0% | NPF | NPF |
| 16 | 38.4°C | 41.0% | NPF | NPF |
| 17 | 38.6°C | 40.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 38.7°C | 40.0% | NPF | NPF |
| 21 | 38.6°C | 39.0% | NPF | NPF |
| 25 | 38.5°C | 39.0% | NPF | NPF |
| 28 | 38.5°C | 40.5% | NPF | NPF |
| 31 | 38.5°C | 41.0% | NPF | NPF |
| 36 | 38.5°C | 41.0% | NPF | NPF |
| 39 | 38.5°C | 41.0% | NPF | NPF |
| 42 | 38.3°C | 42.0% | NPF | NPF |
| 44 | 38.9°C | 42.0% | NPF | NPF |
| 49 | 38.5°C | 41.0% | NPF | T. mutans few |
| 51 | 38.4°C | 40.0% | NPF | T. mutans few |
| 53 | 38.4°C | 40.0% | NPF | NPF |
| 58 | 38.3°C | 41.0% | NPF | NPF |
| 60 | 38.6°C | 41.0% | NPF | NPF |
| 63 | 38.5°C | 40.0% | NPF | NPF |
| 65 | 38.5°C | 40.0% | NPF | NPF |
| 70 | 38.5°C | 41.5% | NPF | NPF |
| 72 | 38.5°C | 41.5% | NPF | NPF |
| 77 | 38.5°C | 41.0% | NPF | NPF |
| 79 | 38.5°C | 40.0% | NPF | NPF |
| 81 | 38.3°C | 40.0% | NPF | NPF |
| 85 | 38.4°C | 40.0% | NPF | NPF |
| 86 | 38.3°C | 41.0% | NPF | NPF |
| 88 | 38.5°C | 41.0% | NPF | T. mutans few |
| 91 | 38.4°C | 40.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.4°C | 39.0% | NPF | NPF |
| 95 | 38.5°C | 39.0% | NPF | NPF |
| 98 | 38.5°C | 40.5% | NPF | NPF |
| 100 | 38.5°C | 40.5% | NPF | NPF |
| 102 | 38.5°C | 40.0% | NPF | NPF |
| 109 | 38.4°C | 40.0% | NPF | NPF |
| 115 | 38.4°C | 41.0% | NPF | NPF |
| 122 | 38.6°C | 41.0% | NPF | NPF |
| 129 | 38.5°C | 42.0% | NPF | T. mutans few |
| 135 | 38.5°C | 41.5% | NPF | T. mutans few |
| 157 | 38.5°C | 40.0% | NPF | NPF |
| 164 | 38.5°C | 41.0% | NPF | NPF |
| 171 | 38.5°C | 40.5% | NPF | NPF |

HEIFER NO. L 15 (CONTROL)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 35.0% | NPF | T. mutans few |
| - 29 | 38.4°C | 35.0% | NPF | T. mutans few |
| - 22 | 38.5°C | 35.5% | NPF | T. mutans few |
| - 15 | 38.5°C | 35.0% | NPF | T. mutans few |
| - 8 | 38.5°C | 35.5% | NPF | NPF |
| - 3 | 38.6°C | 36.0% | NPF | NPF |
| 0 | 38.4°C | 35.5% | NPF | NPF |
| 1 | 38.5°C | 35.5% | NPF | NPF |
| 2 | 38.5°C | 35.0% | NPF | NPF |
| 3 | 38.5°C | 35.0% | NPF | NPF |
| 4 | 38.5°C | 36.5% | NPF | NPF |
| 5 | 38.3°C | 38.0% | NPF | NPF |
| 6 | 38.3°C | 38.0% | NPF | NPF |
| 7 | 38.5°C | 38.0% | NPF | NPF |
| 8 | 38.4°C | 36.5% | NPF | NPF |
| 9 | 38.5°C | 38.0% | NPF | NPF |
| 10 | 38.5°C | 38.5% | NPF | NPF |
| 11 | 38.5°C | 36.5% | NPF | NPF |
| 12 | 38.5°C | 37.0% | NPF | NPF |
| 13 | 38.4°C | 37.0% | NPF | NPF |
| 14 | 38.5°C | 37.0% | NPF | NPF |
| 15 | 38.5°C | 37.5% | NPF | T. mutans few |
| 16 | 38.4°C | 37.0% | NPF | NPF |
| 17 | 38.5°C | 36.5% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 38.4°C | 36.0% | NPF | NPF |
| 21 | 38.5°C | 38.0% | NPF | NPF |
| 25 | 38.6°C | 38.0% | NPF | NPF |
| 28 | 38.7°C | 38.0% | NPF | NPF |
| 31 | 38.6°C | 38.5% | NPF | NPF |
| 36 | 38.6°C | 37.0% | NPF | NPF |
| 39 | 38.6°C | 37.0% | NPF | NPF |
| 42 | 38.5°C | 38.0% | NPF | NPF |
| 44 | 38.5°C | 38.0% | NPF | NPF |
| 49 | 38.6°C | 35.5% | NPF | NPF |
| 51 | 38.5°C | 36.0% | NPF | NPF |
| 53 | 38.5°C | 36.0% | NPF | NPF |
| 58 | 38.4°C | 36.0% | NPF | NPF |
| 60 | 38.5°C | 36.0% | NPF | NPF |
| 63 | 38.5°C | 36.0% | NPF | NPF |
| 65 | 38.3°C | 36.0% | NPF | NPF |
| 70 | 38.5°C | 38.0% | NPF | NPF |
| 72 | 38.5°C | 38.0% | NPF | NPF |
| 77 | 38.4°C | 37.5% | NPF | NPF |
| 79 | 38.6°C | 36.0% | NPF | NPF |
| 81 | 38.5°C | 38.0% | NPF | NPF |
| 85 | 38.5°C | 36.5% | NPF | NPF |
| 86 | 38.5°C | 36.0% | NPF | NPF |
| 88 | 38.5°C | 38.0% | NPF | NPF |
| 91 | 38.5°C | 37.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.6°C | 35.5% | NPF | NPF |
| 95 | 38.5°C | 35.0% | NPF | NPF |
| 98 | 38.7°C | 36.0% | NPF | NPF |
| 100 | 38.5°C | 36.0% | NPF | NPF |
| 102 | 38.5°C | 36.5% | NPF | NPF |
| 109 | 38.5°C | 36.5% | NPF | NPF |
| 115 | 38.4°C | 37.0% | NPF | NPF |
| 122 | 38.5°C | 38.0% | NPF | NPF |
| 129 | 38.5°C | 35.0% | NPF | NPF |
| 135 | 38.5°C | 40.0% | NPF | NPF |
| 157 | 38.6°C | 40.0% | NPF | NPF |
| 164 | 38.4°C | 38.5% | NPF | NPF |
| 171 | 38.5°C | 38.0% | NPF | NPF |

# APPENDIX 2

## CHANGES IN RECTAL TEMPERATURE, PCV AND PARASITEMIA IN PREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH *TRYPANOSOMA VIVAX* DURING THE FIRST TRIMESTER AND THOSE OF THE CONTROLS

HEIFER NO. 1933

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 37.0% | NPF | T. mutans few |
| - 29 | 38.4°C | 38.0% | NPF | T. mutans few |
| - 22 | 38.5°C | 34.0% | NPF | T. mutans few |
| - 15 | 38.5°C | 33.0% | NPF | T. mutans few |
| - 8 | 38.6°C | 43.0% | NPF | T. mutans few |
| - 3 | 38.5°C | 42.0% | NPF | T. mutans few |
| 0 | 38.5°C | 42.0% | NPF | NPF |
| 1 | 38.8°C | 41.6% | NPF | NPF |
| 2 | 38.5°C | 42.0% | NPF | NPF |
| 3 | 38.5°C | 35.0% | Tryps + | T. vivax |
| 4 | 38.5°C | 33.0% | Tryps ++ | T. vivax |
| 5 | 38.5°C | 30.0% | Tryps +++ | T. vivax |
| 6 | 39.0°C | 27.5% | Tryps +++ | T. vivax |
| 7 | 38.8°C | 30.0% | Tryps ++ | T. vivax |
| 8 | 38.5°C | 29.0% | Tryps ++++ | T. vivax |
| 9 | 38.7°C | 28.0% | Tryps ++++ | T. vivax |
| 10 | 38.9°C | 29.0% | Tryps ++++ | T. vivax |
| 11 | 39.0°C | 32.0% | Tryps +++ | T. vivax |
| 12 | 39.2°C | 29.0% | Tryps ++ | T. vivax |
| 13 | 39.5°C | 29.0% | Tryps ++ | T. vivax |
| 14 | 39.5°C | 28.0% | Tryps ++ | T. vivax |
| 15 | 40.0°C | 28.0% | Tryps ++ | T. vivax |
| 16 | 39.1°C | 26.5% | Tryps + | T. vivax |
| 17 | 38.8°C | 28.0% | Tryps ++++ | T. vivax |
| 18 | 38.9°C | 28.0% | Tryps ++++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 21 | 39.1°C | 26.0% | Tryps +++ | T. vivax |
| 25 | 39.5°C | 24.0% | Tryps +++ | T. vivax |
| 28 | 39.0°C | 24.0% | NPF | NPF |
| 31 | 39.1°C | 21.0% | Tryps ++ | T. vivax |
| 36 | 38.7°C | 20.0% | Tryps +++ | T. vivax |
| 39 | 40.0°C | 21.0% | Tryps ++ | T. vivax Aborted |
| 42 | 39.8°C | 28.0% | NPF | NPF |
| 44 | 39.2°C | 24.0% | NPF | NPF |
| 49 | 38.3°C | 26.0% | NPF | NPF |
| 51 | 40.1°C | 27.0% | NPF | T. mutans few |
| 53 | 39.3°C | 27.0% | NPF | T. mutans few |
| 58 | 39.1°C | 30.0% | NPF | T. mutans few |
| 60 | 39.3°C | 28.0% | NPF | NPF |
| 63 | 38.7°C | 28.0% | NPF | NPF |
| 65 | 39.3°C | 28.0% | Tryps ++ | T. vivax |
| 70 | 38.7°C | 24.0% | Tryps + | T. vivax |
| 72 | 39.1°C | 24.0% | NPF | NPF |
| 77 | 38.5°C | 29.0% | NPF | NPF |
| 79 | 38.5°C | 29.0% | NPF | NPF |
| 81 | 38.6°C | 30.0% | NPF | NPF |
| 85 | 38.4°C | 32.0% | NPF | NPF |
| 86 | 38.9°C | 26.0% | NPF | NPF |
| 88 | 38.5°C | 28.0% | NPF | NPF |
| 91 | 38.5°C | 29.0% | NPF | T. mutans few |
| 93 | 38.4°C | 29.0% | NPF | T. mutans few |
| 95 | 38.5°C | 30.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 98 | 38.5°C | 31.0% | NPF | NPF |
| 100 | 38.5°C | 31.5% | NPF | NPF |
| 102 | 38.4°C | 32.0% | NPF | NPF |
| 115 | 38.7°C | 32.5% | NPF | NPF |
| 122 | 38.6°C | 33.0% | NPF | NPF |
| 129 | 38.4°C | 32.0% | NPF | NPF |
| 135 | 38.5°C | 31.0% | NPF | NPF |
| 157 | 38.5°C | 31.0% | NPF | NPF |
| 164 | 38.5°C | 31.0% | NPF | NPF |
| 171 | 38.5°C | 30.0% | NPF | NPF |

HEIFER NO. 1703

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 30.0% | NPF | T. mutans few |
| - 29 | 38.5°C | 31.0% | NPF | T. mutans few |
| - 22 | 38.4°C | 29.5% | NPF | T. mutans few |
| - 15 | 38.5°C | 29.0% | NPF | NPF |
| - 8 | 38.6°C | 29.5% | NPF | NPF |
| - 3 | 38.5°C | 29.0% | NPF | NPF |
| 0 | 38.5°C | 29.0% | NPF | NPF |
| 1 | 38.4°C | 28.5% | NPF | NPF |
| 2 | 38.4°C | 28.5% | NPF | NPF |
| 3 | 38.5°C | 27.0% | NPF | NPF |
| 4 | 38.5°C | 26.0% | Tryps + | NPF |
| 5 | 38.5°C | 24.5% | Tryps ++ | T. vivax |
| 6 | 38.8°C | 25.0% | Tryps ++ | T. vivax |
| 7 | 39.5°C | 25.0% | Tryps ++++ | T. vivax |
| 8 | 39.2°C | 24.5% | Tryps ++++ | T. vivax |
| 9 | 38.5°C | 22.0% | Tryps ++++ | T. vivax |
| 10 | 38.5°C | 23.0% | Tryps ++++ | T. vivax |
| 11 | 38.2°C | 21.0% | Tryps ++++ | T. vivax |
| 12 | 37.7°C | 23.0% | Tryps ++ | T. vivax |
| 13 | 38.5°C | 23.0% | Tryps ++ | T. vivax |
| 14 | 38.8°C | 23.5% | Tryps +++ | T. vivax |
| 15 | 38.9°C | 23.5% | Tryps +++ | T. vivax |
| 16 | 39.9°C | 25.0% | Tryps +++ | T. vivax |
| 17 | 40.0°C | 25.0% | Tryps ++ | T. vivax |
| 18 | 39.9°C | 26.0% | Tryps ++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 21 | 38.8°C | 26.0% | Tryps ++ | T. vivax |
| 25 | 40.0°C | 25.0% | Tryps ++ | T. vivax |
| 28 | 39.5°C | 26.0% | Tryps + | T. vivax |
| 31 | 39.0°C | 24.5% | Tryps + | NPF |
| 36 | 37.6°C | 24.0% | Tryps + | T. vivax |
| 39 | 38.5°C | 24.5% | NPF | NPF |
| 42 | 39.3°C | 25.5% | Tryps + | T. vivax |
| 44 | 38.8°C | 25.5% | Tryps + | T. vivax |
| 49 | 39.8°C | 24.0% | Tryps ++ | T. vivax |
| 51 | 40.2°C | 25.5% | Tryps ++++ | T. vivax |
| 53 | 39.4°C | 20.0% | Tryps +++ | T. vivax |
| 58 | 38.6°C | 28.0% | NPF | NPF |
| 60 | 38.7°C | 28.0% | NPF | NPF |
| 63 | 38.5°C | 24.0% | NPF | NPF |
| 65 | 39.1°C | 27.5% | NPF | NPF |
| 70 | 38.6°C | 21,0% | NPF | NPF |
| 72 | 38.5°C | 24.0% | NPF | NPF |
| 77 | 39.0°C | 26.0% | NPF | NPF |
| 79 | 39.0°C | 27.0% | NPF | NPF |
| 81 | 38.6°C | 29.0% | NPF | NPF |
| 85 | 38.4°C | 29.5% | NPF | NPF |
| 86 | 38.5°C | 28.5% | NPF | NPF |
| 88 | 38.5°C | 30.0% | NPF | NPF |
| 91 | 38.5°C | 30.0% | NPF | NPF |
| 93 | 38.6°C | 29.0% | NPF | NPF |
| 95 | 38.5°C | 29.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 98 | 38.5°C | 31.0% | NPF | NPF |
| 100 | 38.6°C | 30.0% | NPF | NPF |
| 102 | 38.6°C | 30.0% | NPF | NPF |
| 109 | 38.5°C | 30.0% | NPF | NPF |
| 115 | 38.5°C | 28.5% | NPF | NPF |
| 122 | 38.5°C | 29.0% | NPF | NPF |
| 129 | 38.8°C | 27.0% | NPF | NPF |
| 135 | 38.5°C | 28.0% | NPF | NPF |
| 157 | 38.4°C | 27.5% | NPF | NPF |
| 164 | 38.7°C | 30.0% | NPF | NPF |
| 171 | 38.5°C | 29.5% | NPF | NPF |

HEIFER NO. 1714

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.4°C | 42.0% | NPF | T. mutans few |
| - 29 | 38.6°C | 43.0% | NPF | T. mutans few |
| - 22 | 38.5°C | 44.0% | NPF | T. mutans few |
| - 15 | 38.5°C | 44.5% | NPF | NPF |
| - 8 | 38.6°C | 45.0% | NPF | NPF |
| - 3 | 38.6°C | 45.0% | NPF | NPF |
| 0 | 38.5°C | 45.0% | NPF | NPF |
| 1 | 38.5°C | 45.0% | NPF | NPF |
| 2 | 38.4°C | 45.0% | NPF | NPF |
| 3 | 39.0°C | 42.0% | Tryps + | T. vivax |
| 4 | 38.8°C | 34.5% | Tryps ++ | T. vivax |
| 5 | 38.4°C | 35.5% | Tryps ++++ | T. vivax |
| 6 | 38.8°C | 35.0% | Tryps ++++ | T. vivax |
| 7 | 39.0°C | 33.0% | Tryps ++++ | T. vivax |
| 8 | 38.9°C | 33.0% | Tryps +++ | T. vivax |
| 9 | 38.8°C | 33.5% | Tryps +++ | T. vivax |
| 10 | 39.0°C | 35.5% | Tryps +++ | T. vivax |
| 11 | 39.4°C | 34.0% | Tryps +++ | T. vivax |
| 12 | 39.5°C | 34.0% | Tryps ++ | T. vivax |
| 13 | 38.5°C | 31.5% | Tryps + | T. vivax |
| 14 | 37.8°C | 37.5% | Tryps + | T. vivax |
| 15 | 38.7°C | 38.5% | Tryps + | T. vivax |
| 16 | 38.9°C | 34.5% | Tryps ++ | T. vivax |
| 17 | 38.5°C | 35.0% | Tryps + | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 38.8°C | 34.5% | Tryps + | T. vivax |
| 19 | 39.0°C | 30.5% | Tryps + | T. vivax |
| 25 | 38.9°C | 29.5% | Tryps ++ | T. vivax |
| 28 | 38.8°C | 29.0% | Tryps + | T. vivax |
| 31 | 38.6°C | 32.0% | Tryps + | T. vivax |
| 36 | 38.5°C | 34.5% | NPF | NPF |
| 39 | 38.0°C | 37.0% | NPF | NPF |
| 42 | 39.0°C | 34.5% | Tryps + | T. vivax |
| 44 | 38.3°C | 38.0% | NPF | NPF |
| 49 | 38.5°C | 35.5% | NPF | NPF |
| 51 | 39.3°C | 37.0% | NPF | NPF |
| 53 | 39.2°C | 36.5% | NPF | NPF |
| 58 | 39.0°C | 37.0% | NPF | NPF |
| 60 | 39.5°C | 34.0% | NPF | NPF |
| 63 | 38.9°C | 32.0% | Tryps + | T. vivax |
| 65 | 39.5°C | 32.5% | Tryps + | T. vivax |
| 70 | 39.5°C | 32.0% | NPF | NPF |
| 72 | 38.5°C | 32.0% | NPF | NPF |
| 77 | 39.5°C | 30.5% | NPF | NPF |
| 79 | 39.0°C | 32.0% | NPF | NPF |
| 81 | 38.5°C | 32.5% | NPF | NPF |
| 85 | 38,5°C | 33.0% | NPF | NPF |
| 86 | 38.5°C | 34.0% | NPF | NPF |
| 88 | 38.5°C | 33.0% | NPF | NPF |
| 91 | 38,5°C | 33.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.5°C | 33.0% | NPF | NPF |
| 95 | 38.5°C | 34.5% | NPF | NPF |
| 98 | 38.7°C | 35.0% | NPF | NPF |
| 100 | 38.5°C | 33.0% | NPF | NPF |
| 102 | 38.7°C | 32.5% | NPF | NPF |
| 109 | 38.5°C | 32.0% | NPF | NPF |
| 115 | 38.9°C | 33.0% | NPF | NPF |
| 122 | 38.6°C | 36.0% | NPF | NPF |
| 129 | 38.5°C | 35.5% | NPF | NPF |
| 135 | 38.5°C | 33.5% | NPF | NPF |
| 157 | 38.6°C | 33.0% | NPF | NPF |
| 164 | 38.5°C | 33.0% | NPF | NPF |
| 171 | 38.5°C | 34.0% | NPF | NPF |

HEIFER NO. 1817

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 35.0% | NPF | T. mutans few |
| - 29 | 38.7°C | 34.5% | NPF | T. mutans few |
| - 22 | 38.6°C | 34.0% | NPF | T. mutans few |
| - 15 | 38.5°C | 34.0% | NPF | T. mutans few |
| - 8 | 38.5°C | 34.5% | NPF | NPF |
| - 3 | 38.7°C | 35.0% | NPF | NPF |
| 0 | 38.6°C | 34.0% | NPF | NPF |
| 1 | 38.5°C | 34.0% | NPF | NPF |
| 2 | 38.8°C | 35.0% | NPF | NPF |
| 3 | 38.8°C | 32.0% | NPF | NPF |
| 4 | 39.4°C | 30.0% | Tryps + | T. vivax |
| 5 | 39.8°C | 31.0% | Tryps + | T. vivax |
| 6 | 39.2°C | 27.5% | Tryps +++ | T. vivax |
| 7 | 38.6°C | 26.0% | Tryps ++++ | T. vivax |
| 8 | 38.6°C | 25.0% | Tryps ++++ | T. vivax |
| 9 | 38.5°C | 26.0% | Tryps ++++ | T. vivax |
| 10 | 38.8°C | 26.0% | Tryps +++ | T. vivax |
| 11 | 38.9°C | 26.5% | Tryps ++ | T. vivax |
| 12 | 38.4°C | 25.5% | Tryps + | T. vivax |
| 13 | 39.3°C | 26.0% | Tryps + | T. vivax |
| 14 | 39.5°C | 26.0% | Tryps ++ | T. vivax |
| 15 | 38.9°C | 25.0% | Tryps ++ | T. vivax |
| 16 | 38.8°C | 26.0% | Tryps + | T. vivax |
| 17 | 39.0°C | 25.5% | Tryps + | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.9°C | 24.0% | Tryps ++ | T. vivax |
| 21 | 39.8°C | 24.5% | Tryps +++ | T. vivax |
| 25 | 40.0°C | 24.0% | Tryps ++ | T. vivax |
| 28 | 38.7°C | 28.0% | NPF | NPF |
| 31 | 38.5°C | 27.5% | Tryps + | T. vivax |
| 36 | 38.7°C | 26.0% | Tryps +++ | T. vivax |
| 39 | 38.3°C | 28.0% | NPF | NPF |
| 42 | 39.5°C | 28.0% | Tryps + | T. vivax |
| 44 | 39.0°C | 26.0% | Tryps + | T. vivax |
| 49 | 38.6°C | 27.5% | NPF | NPF |
| 51 | 38.9°C | 28.0% | Tryps ++ | T. vivax |
| 53 | 39.0°C | 25.0% | Tryps + | T. vivax |
| 58 | 38.5°C | 28.5% | NPF | NPF |
| 60 | 39.1°C | 24.0% | NPF | NPF |
| 63 | 38.5°C | 22.0% | NPF | NPF |
| 65 | 38.5°C | 25.0% | NPF | NPF |
| 70 | 38.7°C | 24.5% | NPF | NPF |
| 72 | 38.6°C | 26.0% | NPF | NPF |
| 77 | 38.5°C | 25.0% | NPF | NPF |
| 79 | 38.6°C | 26.0% | NPF | NPF |
| 81 | 38.5°C | 25.0% | NPF | NPF |
| 85 | 38.5°C | 27.5% | NPF | NPF |
| 86 | 38.5°C | 27.5% | NPF | NPF |
| 88 | 38.5°C | 27.5% | NPF | NPF |
| 91 | 38.5°C | 29.0% | NPF | T. mutans few |
| 93 | 38.4°C | 29.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 95 | 38.7°C | 28.0% | NPF | NPF |
| 98 | 38.5°C | 27.5% | NPF | NPF |
| 100 | 38.5°C | 27.0% | NPF | NPF |
| 102 | 38.5°C | 27.0% | NPF | NPF |
| 109 | 38.8°C | 28.0% | NPF | NPF |
| 115 | 38.4°C | 29.0% | NPF | NPF |
| 122 | 38.5°C | 28.5% | NPF | NPF |
| 129 | 38.5°C | 27.0% | NPF | NPF |
| 135 | 38.4°C | 28.0% | NPF | NPF |
| 157 | 38.5°C | 27.5% | NPF | NPF |
| 164 | 38.4°C | 27.0% | NPF | NPF |
| 171 | 38.5°C | 27.5% | NPF | NPF |

HEIFER NO. 1764

(Control for 1st Trimester Trial)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 45.0% | NPF | T. mutans few |
| - 29 | 38.5°C | 46.0% | NPF | T. mutans few |
| - 22 | 38.6°C | 46.5% | NPF | T. mutans few |
| - 15 | 38.5°C | 46.0% | NPF | NPF |
| - 8 | 38.5°C | 46.0% | NPF | NPF |
| - 3 | 38.7°C | 46.0% | NPF | NPF |
| 0 | 38.5°C | 47.0% | NPF | NPF |
| 1 | 38.4°C | 46.5% | NPF | NPF |
| 2 | 38.5°C | 46.0% | NPF | NPF |
| 3 | 38.5°C | 47.0% | NPF | NPF |
| 4 | 38.6°C | 46.5% | NPF | NPF |
| 5 | 38.5°C | 46.5% | NPF | NPF |
| 6 | 38.5°C | 46.0% | NPF | NPF |
| 7 | 38.5°C | 48.0% | NPF | NPF |
| 8 | 38.6°C | 46.0% | NPF | NPF |
| 9 | 38.6°C | 45.5% | NPF | NPF |
| 10 | 38.6°C | 44.5% | NPF | NPF |
| 11 | 38.5°C | 44.5% | NPF | NPF |
| 12 | 38.4°C | 44.0% | NPF | NPF |
| 13 | 38.6°C | 44.0% | NPF | T. mutans few |
| 14 | 38.5°C | 47.0% | NPF | T. mutans few |
| 15 | 38.5°C | 47.5% | NPF | T. mutans few |
| 16 | 38.5°C | 46.0% | NPF | T. mutans few |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 17 | 38.5°C | 45.0% | NPF | T. mutans few |
| 18 | 38.5°C | 45.0% | NPF | T. mutans few |
| 21 | 38.5°C | 45.5% | NPF | T. mutans few |
| 25 | 38.5°C | 43.5% | NPF | T. mutans few |
| 28 | 38.4°C | 43.0% | NPF | T. mutans few |
| 31 | 38.5°C | 44.0% | NPF | T. mutans few |
| 36 | 38.6°C | 44.5% | NPF | NPF |
| 39 | 38.5°C | 43.0% | NPF | NPF |
| 42 | 38.5°C | 44.0% | NPF | NPF |
| 44 | 38.5°C | 42.0% | NPF | NPF |
| 49 | 38.5°C | 43.0% | NPF | NPF |
| 51 | 38.5°C | 40.0% | NPF | NPF |
| 53 | 38.5°C | 42.5% | NPF | NPF |
| 58 | 38.5°C | 43.0% | NPF | NPF |
| 60 | 38.6°C | 45.0% | NPF | NPF |
| 63 | 38.6°C | 40.0% | NPF | NPF |
| 65 | 38.5°C | 42.0% | NPF | NPF |
| 70 | 38.5°C | 42.5% | NPF | NPF |
| 72 | 38.4°C | 42.5% | NPF | NPF |
| 77 | 38.5°C | 43.0% | NPF | NPF |
| 79 | 38.5°C | 43.0% | NPF | NPF |
| 81 | 38.6°C | 42.5% | NPF | NPF |
| 85 | 38.4°C | 42.0% | NPF | NPF |
| 86 | 38.6°C | 42.0% | NPF | NPF |
| 88 | 38.5°C | 43.0% | NPF | NPF |
| 91 | 38.5°C | 43.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.4°C | 45.0% | NPF | NPF |
| 95 | 38.6°C | 44.5% | NPF | NPF |
| 98 | 38.5°C | 44.0% | NPF | NPF |
| 100 | 38.5°C | 43.5% | NPF | NPF |
| 102 | 38.5°C | 42.0% | NPF | NPF |
| 109 | 38.5°C | 40.0% | NPF | NPF |
| 115 | 38.5°C | 42.0% | NPF | T. mutans |
| 122 | 38.5°C | 42.0% | NPF | T. mutans |
| 129 | 38.6°C | 43.0% | NPF | T. mutans |
| 135 | 38.7°C | 43.0% | NPF | T. mutans |
| 157 | 38.7°C | 43.0% | NPF | T. mutans |
| 164 | 38.6°C | 44.0% | NPF | T. mutans |
| 171 | 38.5°C | 43.5% | NPF | T. mutans |

HEIFER NO. 1801

(Control for 1st Trimester Trial)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 39.0% | NPF | T. mutans few |
| - 29 | 38.6°C | 35.0% | NPF | T. mutans few |
| - 22 | 38.6°C | 32.0% | NPF | T. mutans few |
| - 15 | 38.6°C | 30.0% | NPF | NPF |
| - 8 | 38.5°C | 32.0% | NPF | NPF |
| - 3 | 38.5°C | 33.0% | NPF | NPF |
| 0 | 38.5°C | 32.5% | NPF | NPF |
| 1 | 38.4°C | 32.5% | NPF | NPF |
| 2 | 38.5°C | 33.0% | NPF | NPF |
| 3 | 38.5°C | 32.5% | NPF | NPF |
| 4 | 38.5°C | 32.5% | NPF | NPF |
| 5 | 38.5°C | 32.0% | NPF | NPF |
| 6 | 38.5°C | 32.0% | NPF | NPF |
| 7 | 38.5°C | 33.0% | NPF | NPF |
| 8 | 38.5°C | 33.0% | NPF | NPF |
| 9 | 38.5°C | 33.5% | NPF | NPF |
| 10 | 38.6°C | 32.5% | NPF | NPF |
| 11 | 38.6°C | 32.5% | NPF | NPF |
| 12 | 38.5°C | 32.0% | NPF | NPF |
| 13 | 38.5°C | 33.0% | NPF | NPF |
| 14 | 38.5°C | 33.0% | NPF | NPF |
| 15 | 38.5°C | 33.5% | NPF | NPF |
| 16 | 38.5°C | 33.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 17 | 38.5°C | 32.5% | NPF | T. mutans few |
| 18 | 38.6°C | 32.0% | NPF | T. mutans few |
| 21 | 38.4°C | 33.0% | NPF | T. mutans few |
| 25 | 38.5°C | 34.0% | NPF | T. mutans few |
| 28 | 38.5°C | 34.5% | NPF | T. mutans few |
| 31 | 38.7°C | 33.5% | NPF | T. mutans few |
| 36 | 38.5°C | 34.0% | NPF | T. mutans few |
| 39 | 38.5°C | 34.0% | NPF | T. mutans few |
| 42 | 38.5°C | 33.5% | NPF | T. mutans few |
| 44 | 38.5°C | 33.0% | NPF | NPF |
| 49 | 38.4°C | 32.5% | NPF | NPF |
| 51 | 38.5°C | 32.0% | NPF | NPF |
| 53 | 38.6°C | 33.0% | NPF | NPF |
| 58 | 38.6°C | 30.5% | NPF | NPF |
| 60 | 38.5°C | 30.5% | NPF | NPF |
| 63 | 38.5°C | 31.0% | NPF | NPF |
| 65 | 38.5°C | 32.5% | NPF | NPF |
| 70 | 38.5°C | 32.0% | NPF | NPF |
| 72 | 38.5°C | 32.0% | NPF | NPF |
| 77 | 38.6°C | 31.5% | NPF | NPF |
| 79 | 38.5°C | 31.0% | NPF | NPF |
| 81 | 38.5°C | 31.0% | NPF | NPF |
| 85 | 38.6°C | 35.0% | NPF | NPF |
| 86 | 38.5°C | 33.0% | NPF | NPF |
| 88 | 38.5°C | 31.0% | NPF | NPF |
| 91 | 38.5°C | 34.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.5°C | 33.5% | NPF | NPF |
| 95 | 38.6°C | 33.0% | NPF | NPF |
| 98 | 38.5°C | 33.0% | NPF | NPF |
| 100 | 38.6°C | 32.5% | NPF | T. mutans few |
| 102 | 38.6°C | 35.0% | NPF | T. mutans few |
| 109 | 38.6°C | 33.5% | NPF | T. mutans few |
| 115 | 38.5°C | 33.0% | NPF | T. mutans few |
| 122 | 38.5°C | 35.0% | NPF | T. mutans few |
| 129 | 38.5°C | 32.5% | NPF | T. mutans few |
| 135 | 38.5°C | 33.0% | NPF | T. mutans few |
| 157 | 38.5°C | 33.5% | NPF | T. mutans few |
| 164 | 38.6°C | 33.0% | NPF | T. mutans few |
| 171 | 38.5°C | 33.0% | NPF | T. mutans few |

APPENDIX 3

CHANGES IN RECTAL TEMPERATURE, PCV AND PARASITEMIA IN PREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH TRYPANOSOMA VIVAX DURING THE SECOND TRIMESTER AND THOSE OF THE CONTROLS

HEIFER NO. 318

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 38.0% | NPF | NPF |
| - 29 | 38.6°C | 37.0% | NPF | NPF |
| - 22 | 38.5°C | 38.5% | NPF | NPF |
| - 15 | 38.5°C | 39.0% | NPF | NPF |
| - 8 | 38.4°C | 36.0% | NPF | NPF |
| - 3 | 38.5°C | 35.5% | NPF | NPF |
| 0 | 38.4°C | 35.0% | NPF | NPF |
| 1 | 38.4°C | 37.0% | NPF | NPF |
| 2 | 38.6°C | 38.0% | NPF | NPF |
| 3 | 38.6°C | 39.5% | NPF | NPF |
| 4 | 38.8°C | 39.0% | NPF | NPF |
| 5 | 38.8°C | 39.0% | NPF | NPF |
| 6 | 38.8°C | 38.5% | NPF | NPF |
| 7 | 38.8°C | 38.0% | Tryps + | T. vivax |
| 8 | 38.6°C | 40.0% | Tryps + | T. vivax |
| 9 | 38.9°C | 37.0% | Tryps ++ | T. vivax |
| 10 | 39.6°C | 39.5% | Tryps +++ | T. vivax |
| 11 | 39.8°C | 36.0% | Tryps +++ | T. vivax |
| 12 | 39.8°C | 36.5% | Tryps +++ | T. vivax |
| 13 | 39.9°C | 35.0% | Tryps +++ | T. vivax |
| 14 | 40.0°C | 35.0% | Tryps +++ | T. vivax |
| 15 | 39.5°C | 34.5% | Tryps +++ | T. vivax |
| 16 | 37.9°C | 31.0% | Tryps ++++ | T. vivax |
| 17 | 38.4°C | 33.0% | Tryps ++++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.8°C | 32.0% | Tryps +++ | T. vivax |
| 21 | 39.8°C | 35.5% | Tryps ++++ | T. vivax |
| 25 | 39.7°C | 33.0% | Tryps +++ | T. vivax |
| 28 | 39.1°C | 31.5% | Tryps +++ | T. vivax |
| 31 | 38.2°C | 30.5% | Tryps ++ | T. vivax |
| 36 | 40.3°C | 32.0% | Tryps ++ | T. vivax |
| 39 | 39.5°C | 31.0% | Tryps ++ | T. vivax |
| 42 | 39.0°C | 30.0% | Tryps + | T. vivax |
| 44 | 39.1°C | 30.0% | Tryps + | T. vivax |
| 49 | 39.0°C | 31.0% | Tryps + | T. vivax |
| 51 | 39.3°C | 32.0% | NPF | NPF |
| 53 | 39.0°C | 30.0% | Tryps + | T. vivax |
| 58 | 39.5°C | 29.5% | Tryps + | T. vivax |
| 60 | 38.5°C | 31.0% | NPF | NPF |
| 63 | 38.6°C | 32.0% | NPF | NPF |
| 65 | 38.4°C | 28.5% | Tryps + | T. vivax |
| 70 | 38.6°C | 29.0% | Tryps + | T. vivax |
| 72 | 38.5°C | 29.5% | NPF | NPF |
| 77 | 38.5°C | 28.0% | NPF | NPF |
| 79 | 38.6°C | 29.0% | NPF | NPF |
| 81 | 38.8°C | 28.0% | NPF | NPF |
| 85 | 40.1°C | 23.5% | NPF | NPF |
| 86 | 38.9°C | 20.5% | NPF | NPF |
| 88 | 38.8°C | 18.0% | Tryps ++ | T. vivax |
| 91 | 38.7°C | 20.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.6 C | 21.0% | NPF | NPF |
| 95 | 38.6 C | 22.5% | NPF | NPF |
| 98 | 38.6 C | 25.5% | NPF | NPF |
| 100 | 38.3 C | 25.0% | NPF | NPF |
| 102 | 28.5 C | 23.5% | NPF | NPF |
| 109 | 38.5 C | 25.0% | NPF | NPF |
| 115 | 38.5 C | 27.0% | NPF | NPF |
| 122 | 38.5 C | 26.5% | NPF | NPF |
| 129 | 38.7 C | 29.5% | NPF | NPF |
| 135 | 38.9 C | 29.0% | NPF | NPF |
| 157 | 38.6 C | 29.5% | NPF | NPF |
| 164 | 38.5 C | 29.0% | NPF | NPF |
| 171 | 38.7 C | 30.0% | NPF | NPF |

HEIFER NO. 327

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 43.0% | NPF | NPF |
| - 29 | 38.4°C | 42.5% | NPF | T. mutans few |
| - 22 | 38.5°C | 42.5% | NPF | T. mutans few |
| - 15 | 38.5°C | 43.0% | NPF | NPF |
| - 8 | 38.6°C | 43.0% | NPF | NPF |
| - 3 | 38.4°C | 42.5% | NPF | NPF |
| 0 | 38.5°C | 43.0% | NPF | NPF |
| 1 | 38.5°C | 42.0% | NPF | NPF |
| 2 | 38.8°C | 40.0% | NPF | NPF |
| 3 | 37.9°C | 43.0% | Tryps + | NPF |
| 4 | 38.4°C | 35.5% | Tryps ++ | T. vivax |
| 5 | 38.8°C | 38.0% | Tryps ++ | T. vivax |
| 6 | 38.8°C | 38.0% | Tryps +++ | T. vivax |
| 7 | 39.4°C | 37.5% | Tryps ++++ | T. vivax |
| 8 | 39.2°C | 34.0% | Tryps ++++ | T. vivax |
| 9 | 39.1°C | 34.0% | Tryps ++++ | T. vivax |
| 10 | 39.4°C | 33.0% | Tryps ++++ | T. vivax |
| 11 | 39.0°C | 33.5% | Tryps ++++ | T. vivax |
| 12 | 39.0°C | 30.5% | Tryps ++++ | T. vivax |
| 13 | 39.4°C | 30.5% | Tryps +++ | T. vivax |
| 14 | 39.5°C | 30.5% | Tryps ++ | T. vivax |
| 15 | 39.5°C | 28.5% | Tryps ++ | T. vivax |
| 16 | 40.3°C | 30.0% | Tryps ++ | T. vivax |
| 17 | 39.5°C | 30.0% | Tryps ++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.5°C | 30.0% | Tryps ++ | T. vivax |
| 21 | 39.8°C | 30.5% | Tryps + | T. vivax |
| 25 | 39.0°C | 30.0% | Tryps + | T. vivax |
| 28 | 39.4°C | 28.5% | Tryps + | T. vivax |
| 31 | 39.4°C | 26.0% | NPF | NPF |
| 36 | 39.6°C | 30.0% | Tryps + | T. vivax |
| 39 | 39.2°C | 30.0% | Tryps + | T. vivax |
| 42 | 39.0°C | 30.0% | NPF | NPF |
| 44 | 39.5°C | 28.0% | Tryps + | T. vivax |
| 49 | 39.5°C | 29.0% | NPF | NPF |
| 51 | 39.4°C | 30.0% | NPF | NPF |
| 53 | 39.4°C | 28.0% | Tryps ++ | T. vivax |
| 58 | 39.0°C | 26.0% | Tryps + | T. vivax |
| 60 | 38.5°C | 29.0% | Tryps + | T. vivax |
| 63 | 38.5°C | 30.0% | Tryps + | T. vivax |
| 65 | 38.0°C | 30.0% | NPF | NPF |
| 70 | 38.5°C | 30.0% | Tryps + | T. vivax |
| 72 | 38.5°C | 31.0% | Tryps + | T. vivax |
| 77 | 38.6°C | 30.0% | NPF | NPF |
| 79 | 38.4°C | 30.0% | Tryps + | NPF |
| 81 | 38.5°C | 30.0% | Tryps ++ | T. vivax |
| 85 | 38.8°C | 30.0% | NPF | NPF |
| 86 | 38.5°C | 29.5% | NPF | NPF |
| 88 | 38.4°C | 27.0% | NPF | NPF |
| 91 | 38.6°C | 28.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.6°C | 30.0% | NPF | NPF |
| 95 | 38.5°C | 29.0% | NPF | NPF |
| 98 | 38.5°C | 26.0% | NPF | NPF |
| 100 | 38.4°C | 24.5% | Tryps + | T. vivax |
| 102 | 38.4°C | 25.0% | NPF | NPF |
| 109 | 38.5°C | 27.0% | NPF | NPF |
| 115 | 38.5°C | 26.0% | NPF | NPF |
| 122 | 38.5°C | 30.0% | NPF | NPF |
| 129 | 38.5°C | 30.0% | NPF | NPF |
| 133 | 38.6°C | 31.0% | NPF | NPF |
| 157 | 38.5°C | 30.5% | NPF | NPF |
| 164 | 38.5°C | 30.0% | NPF | NPF |
| 171 | 38.5°C | 30.0% | NPF | NPF |

HEIFER NO. 325

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 40.0% | NPF | NPF |
| - 29 | 38.4°C | 40.0% | NPF | NPF |
| - 22 | 38.5°C | 39.5% | NPF | T. mutans few |
| - 15 | 38.5°C | 40.0% | NPF | T. mutans few |
| - 8 | 38.6°C | 38.5% | NPF | T. mutans few |
| - 3 | 38.5°C | 40.5% | NPF | NPF |
| 0 | 38.4°C | 40.0% | NPF | NPF |
| 1 | 38.5°C | 40.0% | NPF | NPF |
| 2 | 38.7°C | 38.5% | NPF | NPF |
| 3 | 38.5°C | 39.5% | NPF | NPF |
| 4 | 38.6°C | 40.0% | NPF | NPF |
| 5 | 38.5°C | 39.0% | NPF | NPF |
| 6 | 38.4°C | 39.0% | NPF | NPF |
| 7 | 38.8°C | 39.0% | NPF | NPF |
| 8 | 39.0°C | 39.0% | Tryps + | T. vivax |
| 9 | 38.9°C | 38.5% | Tryps ++ | T. vivax |
| 10 | 39.5°C | 38.5% | Tryps ++ | T. vivax |
| 11 | 40.0°C | 37.0% | Tryps ++++ | T. vivax |
| 12 | 39.5°C | 37.5% | Tryps ++++ | T. vivax |
| 13 | 39.0°C | 36.0% | Tryps ++++ | T. vivax |
| 14 | 38.8°C | 36.0% | Tryps ++++ | T. vivax |
| 15 | 39.0°C | 34.0% | Tryps ++++ | T. vivax |
| 16 | 39.5°C | 30.0% | Tryps ++++ | T. vivax |
| 17 | 39.4°C | 30.0% | Tryps +++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.2°C | 30.0% | Tryps ++ | T. vivax |
| 21 | 39.0°C | 29.5% | Tryps +++ | T. vivax |
| 25 | 38.0°C | 26.5% | Tryps ++ | T. vivax |
| 28 | 38.5°C | 30.5% | Tryps ++ | T. vivax |
| 31 | 38.8°C | 27.0% | Tryps + | T. vivax |
| 36 | 38.9°C | 26.0% | Tryps ++ | T. vivax |
| 39 | 39.2°C | 26.0% | Tryps ++ | T. vivax |
| 42 | 39.0°C | 26.0% | Tryps + | T. vivax |
| 44 | 38.8°C | 24.5% | NPF | NPF |
| 49 | 38.7°C | 23.5% | Tryps + | NPF |
| 51 | 38.0°C | 24.0% | NPF | NPF |
| 53 | 38.8°C | 24.0% | NPF | NPF |
| 58 | 39.1°C | 22.0% | Tryps ++ | T. vivax |
| 60 | 39.4°C | 22.0% | Tryps + | T. vivax |
| 63 | 39.6°C | 22.0% | NPF | NPF |
| 65 | 39.7°C | 24.5% | Tryps + | T. vivax |
| 70 | 39.8°C | 25.0% | NPF | NPF |
| 72 | 38.4°C | 22.0% | Tryps + | NPF |
| 77 | 38.8°C | 16.0% | Tryps + | T. vivax |
| 79 | 38.5°C | 17.0% | Tryps + | NPF |
| 81 | 38.4°C | 20.5% | NPF | NPF |
| 85 | 35.5°C | 20.0% | NPF | NPF |
| 86 | 38.5°C | 20.0% | NPF | NPF |
| 88 | 38.5°C | 21.0% | NPF | NPF |
| 91 | 38.5°C | 24.0% | NPF | NPF |

HEIFER NO. 337

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 35.0% | NPF | NPF |
| - 29 | 38.5°C | 35.0% | NPF | NPF |
| - 22 | 38.6°C | 34.5% | NPF | NPF |
| - 15 | 38.5°C | 35.0% | NPF | T. mutans few |
| - 8 | 38.5°C | 35.5% | NPF | T. mutans few |
| - 3 | 38.4°C | 35.0% | NPF | NPF |
| 0 | 38.4°C | 35.0% | NPF | NPF |
| 1 | 38.5°C | 35.0% | NPF | NPF |
| 2 | 38.5°C | 35.5% | NPF | NPF |
| 3 | 38.5°C | 35.0% | NPF | NPF |
| 4 | 39.7°C | 34.5% | Tryps + | T. vivax |
| 5 | 39.5°C | 34.0% | Tryps +++ | T. vivax |
| 6 | 39.6°C | 34.0% | Tryps +++ | T. vivax |
| 7 | 39.8°C | 33.0% | Tryps ++++ | T. vivax |
| 8 | 39.9°C | 32.5% | Tryps ++++ | T. vivax |
| 9 | 38.5°C | 30.0% | Tryps ++ | T. vivax |
| 10 | 39.4°C | 28.5% | Tryps +++ | T. vivax |
| 11 | 39.5°C | 27.5% | Tryps +++ | T. vivax |
| 12 | 39.5°C | 27.5% | Tryps ++++ | T. vivax |
| 13 | 39.4°C | 27.5% | Tryps ++++ | T. vivax |
| 14 | 39.5°C | 27.0% | Tryps ++++ | T. vivax |
| 15 | 39.8°C | 27.0% | Tryps ++++ | T. vivax |
| 16 | 39.5°C | 25.0% | Tryps ++++ | T. vivax |
| 17 | 39.4°C | 25.5% | Tryps ++++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.0°C | 27.0% | Tryps +++ | T. vivax |
| 21 | 39.5°C | 27.0% | Tryps ++ | T. vivax |
| 25 | 38.7°C | 28.5% | Tryps +++ | T. vivax |
| 28 | 39.0°C | 28.0% | Tryps + | T. vivax |
| 31 | 39.0°C | 28.5% | Tryps + | T. vivax |
| 36 | 38.6°C | 25.0% | NPF | NPF |
| 39 | 39.0°C | 25.0% | Tryps + | T. vivax |
| 42 | 39.0°C | 25.0% | NPF | NPF |
| 44 | 39.5°C | 27.0% | Tryps + | T. vivax |
| 49 | 39.0°C | 22.0% | Tryps + | T. vivax |
| 51 | 39.1°C | 21.5% | NPF | NPF |
| 53 | 39.2°C | 19.0% | NPF | NPF |
| 58 | 39.5°C | 17.0% | NPF | NPF |
| 60 | 38.4°C | 21.0% | Tryps + | T. vivax |
| 63 | 38.5°C | 24.0% | NPF | NPF |
| 65 | 38.5°C | 26.0% | NPF | NPF |
| 70 | 38.5°C | 26.0% | NPF | NPF |
| 72 | 38.8°C | 28.5% | NPF | NPF |
| 77 | 38.5°C | 28.5% | NPF | NPF |
| 79 | 38.9°C | 28.5% | NPF | NPF |
| 81 | 38.5°C | 29.0% | NPF | NPF |
| 85 | 38.4°C | 27.0% | NPF | NPF |
| 86 | 38.5°C | 27.5% | NPF | NPF |
| 88 | 38.5°C | 28.0% | NPF | NPF |
| 91 | 38.7°C | 27.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.5°C | 25.0% | NPF | NPF |
| 95 | 38.4°C | 27.5% | NPF | NPF |
| 98 | 38.6°C | 28.0% | NPF | NPF |
| 100 | 38.5°C | 28.0% | NPF | NPF |
| 102 | 38.5°C | 28.0% | NPF | NPF |
| 109 | 38.3°C | 27.5% | NPF | NPF |
| 115 | 37.8°C | 28.0% | NPF | NPF |
| 122 | 38.5°C | 28.5% | NPF | NPF |
| 129 | 38.3°C | 29.0% | NPF | NPF |
| 135 | 38.5°C | 29.0% | NPF | NPF |
| 157 | 38.5°C | 31.5% | NPF | NPF |
| 164 | 38.5°C | 31.0% | NPF | NPF |
| 171 | 38.5°C | 30.0% | NPF | NPF |

HEIFER NO. L 39

(Control Animal)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.6°C | 33.0% | NPF | T. mutans few |
| - 29 | 38.6°C | 32.0% | NPF | T. mutans few |
| - 22 | 38.5°C | 32.5% | NPF | T. mutans few |
| - 15 | 38.7°C | 32.5% | NPF | NPF |
| - 8 | 38.5°C | 32.5% | NPF | NPF |
| - 3 | 38.5°C | 32.5% | NPF | NPF |
| 0 | 38.4°C | 32.5% | NPF | NPF |
| 1 | 38.5°C | 32.0% | NPF | NPF |
| 2 | 38.5°C | 32.0% | NPF | NPF |
| 3 | 38.6°C | 32.5% | NPF | NPF |
| 4 | 38.4°C | 33.0% | NPF | NPF |
| 5 | 38.7°C | 32.5% | NPF | NPF |
| 6 | 38.5°C | 32.5% | NPF | NPF |
| 7 | 38.6°C | 32.5% | NPF | NPF |
| 8 | 38.3°C | 32.5% | NPF | NPF |
| 9 | 38.5°C | 33.0% | NPF | NPF |
| 10 | 38.5°C | 32.0% | NPF | T. mutans few |
| 11 | 38.4°C | 32.0% | NPF | NPF |
| 12 | 38.5°C | 32.0% | NPF | NPF |
| 13 | 38.5°C | 33.0% | NPF | NPF |
| 14 | 38.7°C | 33.0% | NPF | NPF |
| 15 | 38.6°C | 32.5% | NPF | T. mutans few |
| 16 | 38.3°C | 32.5% | NPF | T. mutans few |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 17 | 38.5°C | 32.0% | NPF | NPF |
| 18 | 38.6°C | 31.5% | NPF | NPF |
| 21 | 38.4°C | 31.5% | NPF | NPF |
| 25 | 38.4°C | 31.5% | NPF | NPF |
| 28 | 38.6°C | 31.0% | NPF | NPF |
| 31 | 38.5°C | 30.0% | NPF | NPF |
| 36 | 38.5°C | 23.0% | NPF | NPF |
| 39 | 38.5°C | 32.5% | NPF | NPF |
| 42 | 38.5°C | 30.5% | NPF | NPF |
| 44 | 38.5°C | 31.0% | NPF | NPF |
| 49 | 38.5°C | 31.5% | NPF | NPF |
| 51 | 38.7°C | 32.0% | NPF | NPF |
| 53 | 38.6°C | 32.0% | NPF | NPF |
| 58 | 38.3°C | 32.0% | NPF | NPF |
| 60 | 37.0°C | 32.0% | NPF | NPF |
| 63 | 38.5°C | 33.0% | NPF | NPF |
| 65 | 38.5°C | 33.0% | NPF | NPF |
| 70 | 38.6°C | 33.0% | NPF | NPF |
| 72 | 38.7°C | 32.5% | NPF | NPF |
| 77 | 38.6°C | 32.0% | NPF | NPF |
| 79 | 38.5°C | 32.0% | NPF | NPF |
| 81 | 38.5°C | 32.0% | NPF | NPF |
| 85 | 38.5°C | 32.0% | NPF | NPF |
| 86 | 38.5°C | 32.0% | NPF | NPF |
| 91 | 38.3°C | 32.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.5°C | 33.5% | NPF | NPF |
| 95 | 38.5°C | 33.5% | NPF | NPF |
| 98 | 38.4°C | 31.0% | NPF | NPF |
| 100 | 38.5°C | 30.0% | NPF | NPF |
| 102 | 38.5°C | 32.5% | NPF | NPF |
| 109 | 38.4°C | 32.0% | NPF | NPF |
| 115 | 38.6°C | 30.0% | NPF | NPF |
| 129 | 38.5°C | 30.0% | NPF | NPF |
| 135 | 38.5°C | 30.0% | NPF | NPF |
| 157 | 38.5°C | 31.0% | NPF | NPF |
| 164 | 38.5°C | 31.5% | NPF | NPF |
| 171 | 38.3°C | 32.0% | NPF | NPF |

HEIFER NO. 227

(Control Animal)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.6°C | 40.0% | NPF | NPF |
| - 29 | 38.5°C | 40.0% | NPF | T. mutans few |
| - 22 | 38.5°C | 41.0% | NPF | T. mutans few |
| - 15 | 38.4°C | 39.0% | NPF | T. mutans few |
| - 8 | 38.5°C | 39.0% | NPF | T. mutans few |
| - 3 | 38.5°C | 39.0% | NPF | NPF |
| 0 | 38.5°C | 39.5% | NPF | NPF |
| 1 | 38.5°C | 39.5% | NPF | NPF |
| 2 | 38.5°C | 39.5% | NPF | NPF |
| 3 | 38.5°C | 39.0% | NPF | NPF |
| 4 | 38.4°C | 41.0% | NPF | NPF |
| 5 | 38.4°C | 39.0% | NPF | NPF |
| 6 | 38.3°C | 43.0% | NPF | NPF |
| 7 | 38.6°C | 42.5% | NPF | NPF |
| 8 | 38.5°C | 42.5% | NPF | NPF |
| 9 | 38.5°C | 40.0% | NPF | NPF |
| 10 | 38.7°C | 40.0% | NPF | NPF |
| 11 | 38.5°C | 40.0% | NPF | NPF |
| 12 | 38.6°C | 40.0% | NPF | NPF |
| 13 | 38.4°C | 42.0% | NPF | NPF |
| 14 | 38.5°C | 41.0% | NPF | NPF |
| 15 | 38.5°C | 40.0% | NPF | NPF |
| 16 | 38.5°C | 40.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 17 | 38.4°C | 40.0% | NPF | NPF |
| 18 | 38.5°C | 39.0% | NPF | NPF |
| 21 | 38.5°C | 39.5% | NPF | NPF |
| 25 | 38.6°C | 39.5% | NPF | NPF |
| 28 | 38.4°C | 38.0% | NPF | NPF |
| 31 | 38.5°C | 40.0% | NPF | NPF |
| 36 | 38.5°C | 39.5% | NPF | NPF |
| 39 | 38.5°C | 38.0% | NPF | NPF |
| 42 | 38.5°C | 38.0% | NPF | NPF |
| 44 | 38.5°C | 39.5% | NPF | NPF |
| 49 | 38.5°C | 39.5% | NPF | NPF |
| 51 | 38.7°C | 39.5% | NPF | NPF |
| 53 | 38.5°C | 39.5% | NPF | NPF |
| 58 | 38.4°C | 59.5% | NPF | NPF |
| 60 | 38.5°C | 39.5% | NPF | NPF |
| 63 | 38.6°C | 40.5% | NPF | NPF |
| 65 | 38.3°C | 39.5% | NPF | NPF |
| 70 | 38.5°C | 41.0% | NPF | NPF |
| 72 | 38.4°C | 40.0% | NPF | NPF |
| 77 | 38.7°C | 39.5% | NPF | NPF |
| 79 | 38.5°C | 40.0% | NPF | NPF |
| 81 | 38.5°C | 38.5% | NPF | NPF |
| 85 | 38.5°C | 39.0% | NPF | NPF |
| 86 | 38.5°C | 39.0% | NPF | NPF |
| 88 | 38.5°C | 39.5% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 91 | 38.5°C | 40.0% | NPF | NPF |
| 93 | 38.5°C | 43.0% | NPF | NPF |
| 95 | 38.4°C | 42.0% | NPF | NPF |
| 98 | 38.6°C | 41.5% | NPF | NPF |
| 100 | 38.5°C | 42.5% | NPF | NPF |
| 102 | 38.5°C | 39.0% | NPF | NPF |
| 109 | 38.5°C | 38.5% | NPF | NPF |
| 115 | 38.5°C | 39.0% | NPF | T. mutans few |
| 122 | 38.5°C | 39.5% | NPF | T. mutans few |
| 129 | 38.5°C | 39.5% | NPF | NPF |
| 135 | 38.5°C | 40.0% | NPF | NPF |
| 157 | 38.4°C | 41.0% | NPF | NPF |
| 164 | 38.5°C | 42.0% | NPF | NPF |
| 171 | 38.5°C | 40.0% | NPF | NPF |

APPENDIX 4

CHANGES IN RECTAL TEMPERATURE, PCV AND PARASITEMIA IN PREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH TRYPANOSOMA VIVAX DURING THE THIRD TRIMESTER AND THOSE OF THE CONTROLS

HEIFER NO. 1708

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 49.0% | NPF | T. mutans few |
| - 29 | 38.5°C | 47.0% | NPF | T. mutans few |
| - 22 | 38.6°C | 43.0% | NPF | T. mutans few |
| - 15 | 38.5°C | 43.0% | NPF | NPF |
| - 8 | 38.5°C | 43.0% | NPF | NPF |
| - 3 | 38.5°C | 42.5% | NPF | NPF |
| 0 | 38.5°C | 42.5% | NPF | NPF |
| 1 | 38.5°C | 41.5% | NPF | NPF |
| 2 | 39.5°C | 40.5% | Tryps + | NPF |
| 3 | 39.8°C | 37.0% | Tryps + | NPF |
| 4 | 40.0°C | 35.0% | Tryps ++ | T. vivax |
| 5 | 39.7°C | 33.5% | Tryps +++ | T. vivax |
| 6 | 39.5°C | 26.5% | Tryps ++++ | T. vivax |
| 7 | 39.4°C | 26.0% | Tryps ++++ | T. vivax |
| 8 | 39.4°C | 26.0% | Tryps ++++ | T. vivax |
| 9 | 39.5°C | 28.0% | Tryps ++++ | T. vivax |
| 10 | 39.5°C | 27.5% | Tryps ++++ | T. vivax |
| 11 | 39.5°C | 28.0% | Tryps ++++ | T. vivax |
| 12 | 39.6°C | 26.0% | Tryps +++ | T. vivax |
| 13 | 39.5°C | 25.5% | Tryps +++ | T. vivax |
| 14 | 39.3°C | 25.5% | Tryps +++ | T. vivax |
| 15 | 39.7°C | 25.0% | Tryps +++ | T. vivax |
| 16 | 39.2°C | 16.5% | Tryps + | T. vivax |
| 17 | 39.0°C | 13.0% | Tryps ++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.5°C | 12.5% | Tryps + | NPF |
| 21 | 39.6°C | 14.0% | Tryps + | T. vivax |
| 25 | 39.9°C | 17.5% | Tryps + | T. vivax |
| 28 | 39.4°C | 18.5% | Tryps + | T. vivax |
| 31 | 39.0°C | 20.0% | Tryps + | T. vivax |
| 36 | 39.5°C | 25.5% | Tryps + | T. vivax |
| 39 | 39.9°C | 25.5% | Tryps + | T. vivax |
| 40 | 40.0°C | 25.5% | Tryps ++ | T. vivax |
| 42 | 39.2°C | 25.5% | Tryps + | T. vivax |
| 44 | 38.0°C | 19.5% | Tryps + | T. vivax |
| 49 | 39.5°C | 25.0% | NPF | NPF |
| 51 | 39.6°C | 26.0% | NPF | NPF |
| 53 | 39.1°C | 29.0% | NPF | NPF |
| 58 | 39.6°C | 26.0% | Tryps + | NPF |
| 60 | 39.5°C | 24.5% | Tryps + | T. vivax |
| 63 | 39.6°C | 28.0% | NPF | NPF |
| 65 | 39.0°C | 25.0% | NPF | NPF |
| 70 | 38.4°C | 22.0% | NPF | NPF |
| 72 | 38.5°C | 26.5% | NPF | NPF |
| 77 | 38.6°C | 25.5% | NPF | NPF |
| 79 | 39.0°C | 24.0% | NPF | NPF |
| 81 | 39.0°C | 24.0% | NPF | NPF |
| 85 | 38.8°C | 25.5% | NPF | NPF |
| 86 | 38.5°C | 26.5% | NPF | NPF |
| 88 | 38.5°C | 25.5% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 91 | 38.5°C | 30.0% | NPF | NPF |
| 93 | 39.5°C | 30.5% | NPF | NPF |
| 95 | 39.6°C | 25.0% | NPF | NPF |
| 98 | 38.9°C | 28.0% | NPF | NPF |
| 100 | 38.5°C | 28.5% | NPF | NPF |
| 102 | 38.5°C | 29.0% | NPF | NPF |
| 109 | 38.5°C | 29.5% | NPF | NPF |
| 115 | 38.5°C | 29.0% | NPF | NPF |
| 122 | 38.5°C | 30.0% | NPF | NPF |
| 129 | 38.7°C | 31.0% | NPF | NPF |
| 135 | 38.5°C | 30.0% | NPF | NPF |
| 157 | 38.6°C | 31.5% | NPF | NPF |
| 164 | 38.5°C | 31.0% | NPF | NPF |
| 171 | 38.6°C | 30.0% | NPF | NPF |

HEIFER NO. 1856

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 38.5% | NPF | NPF |
| - 29 | 38.5°C | 35.5% | NPF | NPF |
| - 22 | 38.5°C | 34.0% | NPF | NPF |
| - 15 | 38.6°C | 37.0% | NPF | NPF |
| - 8 | 38.5°C | 37.5% | NPF | NPF |
| - 3 | 38.5°C | 37.5% | NPF | NPF |
| 0 | 38.5°C | 37.5% | NPF | NPF |
| 1 | 39.3°C | 38.0% | NPF | NPF |
| 3 | 39.5°C | 35.0% | NPF | NPF |
| 4 | 39.6°C | 34.0% | Tryps + | T. vivax |
| 5 | 39.6°C | 33.0% | Tryps + | T. vivax |
| 6 | 39.7°C | 34.0% | Tryps ++ | T. vivax |
| 7 | 39.8°C | 33.5% | Tryps ++++ | T. vivax |
| 8 | 39.9°C | 34.0% | Tryps ++++ | T. vivax |
| 9 | 39.9°C | 34.5% | Tryps ++++ | T. vivax |
| 10 | 38.8°C | 33.0% | Tryps ++++ | T. vivax |
| 11 | 38.9°C | 31.0% | Tryps ++++ | T. vivax |
| 12 | 40.0°C | 30.0% | Tryps ++++ | T. vivax |
| 13 | 39.5°C | 29.0% | Tryps ++++ | T. vivax |
| 14 | 39.9°C | 28.0% | Tryps ++++ | T. vivax |
| 15 | 39.9°C | 25.5% | Tryps ++++ | T. vivax |
| 16 | 39.2°C | 28.0% | Tryps ++ | T. vivax |
| 17 | 39.5°C | 28.5% | Tryps +++ | T. vivax |
| 18 | 39.9°C | 27.5% | Tryps ++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 21 | 39.9°C | 28.5% | Tryps ++ | T. vivax |
| 23 | 40.0°C | 29.0% | Tryps ++ | T. vivax |
| 25 | 39.5°C | 29.5% | Tryps + | T. vivax |
| 28 | 38.8°C | 30.5% | Tryps + | T. vivax |
| 31 | 39.0°C | 35.6% | NPF | NPF |
| 36 | 38.8°C | 33.5% | Tryps + | T. vivax |
| 39 | 38.8°C | 33.0% | NPF | NPF |
| 42 | 38.7°C | 37.0% | NPF | NPF |
| 44 | 38.5°C | 32.0% | NPF | NPF |
| 49 | 38.8°C | 34.5% | Tryps + | T. vivax |
| 51 | 38.6°C | 30.5% | Tryps + | T. vivax |
| 53 | 38.5°C | 31.5% | Tryps + | NPF |
| 58 | 38.5°C | 30.0% | NPF | NPF |
| 60 | 39.0°C | 33.0% | Tryps + | T. vivax |
| 63 | 39.0°C | 33.0% | Tryps + | T. vivax |
| 65 | 38.8°C | 26.0% | Tryps + | NPF |
| 70 | 38.8°C | 27.0% | NPF | NPF |
| 72 | 38.7°C | 27.5% | NPF | NPF |
| 77 | 39.0°C | 28.0% | NPF | NPF |
| 79 | 39.2°C | 32.0% | NPF | NPF |
| 81 | 39.2°C | 31.5% | NPF | NPF |
| 85 | 39.0°C | 30.5% | NPF | NPF |
| 86 | 38.8°C | 31.0% | NPF | NPF |
| 88 | 38.5°C | 31.0% | NPF | NPF |
| 91 | 38.9°C | 33.0% | Tryps + | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.9°C | 33.0% | Tryps + | T. vivax |
| 95 | 38.7°C | 26.0% | Tryps + | T. vivax |
| 98 | 38.5°C | 27.0% | NPF | NPF |
| 100 | 38.5°C | 28.5% | NPF | NPF |
| 102 | 38.6°C | 32.0% | NPF | NPF |
| 109 | 38.5°C | 31.0% | NPF | NPF |
| 115 | 38.5°C | 31.0% | NPF | NPF |
| 122 | 38.5°C | 30.5% | NPF | NPF |

HEIFER NO. 1592

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 37.5% | NPF | T. mutans few |
| - 29 | 38.5°C | 37.5% | NPF | T. mutans few |
| - 22 | 38.5°C | 37.0% | NPF | NPF |
| - 15 | 38.6°C | 37.0% | NPF | NPF |
| - 8 | 38.5°C | 37.0% | NPF | NPF |
| - 3 | 38.5°C | 36.5% | NPF | NPF |
| 0 | 38.4°C | 36.5% | NPF | NPF |
| 1 | 38.5°C | 36.5% | NPF | NPF |
| 2 | 38.8°C | 38.5% | NPF | NPF |
| 3 | 38.9°C | 32.0% | NPF | NPF |
| 4 | 39.4°C | 32.0% | NPF | NPF |
| 5 | 39.6°C | 33.0% | Tryps + | NPF |
| 6 | 40.0°C | 27.5% | Tryps + | T. vivax |
| 7 | 40.3°C | 25.0% | Tryps ++ | T. vivax |
| 8 | 40.2°C | 25.0% | Tryps +++ | T. vivax |
| 9 | 40,0°C | 24.0% | Tryps ++++ | T. vivax |
| 10 | 40,0°C | 23,0% | Tryps ++++ | T. vivax |
| 11 | 39.9°C | 22.5% | Tryps ++++ | T. vivax |
| 12 | 40,0°C | 22,0% | Tryps ++++ | T. vivax |
| 13 | 40.0°C | 19.5% | Tryps ++++ | T, vivax |
| 14 | 39.4°C | 20.0% | Tryps +++ | T. vivax |
| 15 | 39.8°C | 25.0% | Tryps ++++ | T. vivax |
| 16 | 39.9°C | 23.0% | Tryps +++ | T. vivax |
| 17 | 39.8°C | 28.0% | Tryps +++ | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.9°C | 27.5% | Tryps +++ | T. vivax |
| 21 | 39.5°C | 30.0% | Tryps + | T. vivax |
| 25 | 39.6°C | 31.0% | Tryps + | T. vivax |
| 28 | 39.4°C | 30.0% | Tryps + | T. vivax |
| 31 | 38.7°C | 27.0% | Tryps + | T. vivax |
| 36 | 38.7°C | 26.5% | Tryps + | T. vivax |
| 39 | 39.7°C | 27.0% | Tryps + | T. vivax |
| 42 | 38.5°C | 27.5% | Tryps + | T. vivax |
| 44 | 38.5°C | 27.5% | NPF | NPF |
| 49 | 38.5°C | 28.0% | Tryps + | T. vivax |
| 51 | 38.7°C | 29.5% | Tryps + | T. vivax |
| 53 | 38.5°C | 28.5% | NPF | NPF |
| 58 | 39.2°C | 26.0% | Tryps + | T. vivax |
| 60 | 38.7°C | 26.0% | Tryps + | T. vivax |
| 63 | 38.9°C | 28.0% | Tryps + | T. vivax |
| 65 | 38.5°C | 16.5% | Tryps + | T. vivax |
| 70 | 38.6°C | 15.0% | NPF | NPF |
| 72 | 38.5°C | 15.0% | NPF | NPF |
| 77 | 38.5°C | 14.0% | Tryps + | T. vivax |
| 79 | 39.5°C | 20.0% | NPF | NPF |
| 81 | 38.5°C | 28.0% | NPF | NPF |
| 85 | 38.6°C | 26.0% | NPF | NPF |
| 86 | 38.4°C | 28.0% | NPF | NPF |
| 88 | 38.5°C | 28.0% | NPF | NPF |
| 91 | 38.4°C | 28.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.5°C | 28.5% | NPF | NPF |
| 95 | 38.5°C | 28.0% | NPF | NPF |
| 98 | 38.5°C | 28.5% | NPF | NPF |
| 100 | 38.5°C | 29.0% | NPF | NPF |
| 102 | 38.6°C | 28.5% | NPF | NPF |
| 109 | 38.5°C | 27.0% | NPF | NPF |
| 115 | 38.5°C | 29.0% | NPF | NPF |
| 122 | 38.5°C | 30.0% | NPF | NPF |

HEIFER NO. 402

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 32.0% | NPF | NPF |
| - 29 | 38.5°C | 32.0% | NPF | NPF |
| - 22 | 38.6°C | 30.5% | NPF | NPF |
| - 15 | 38.5°C | 31.0% | NPF | NPF |
| - 8 | 38.5°C | 31.5% | NPF | NPF |
| - 3 | 38.5°C | 32.0% | NPF | NPF |
| 0 | 38.5°C | 32.0% | NPF | NPF |
| 1 | 38.5°C | 31.5% | NPF | NPF |
| 2 | 38.8°C | 30.0% | NPF | NPF |
| 3 | 39.2°C | 32.0% | Tryps + | NPF |
| 4 | 39.4°C | 30.0% | Tryps ++++ | T. vivax |
| 5 | 39.4°C | 30.0% | Tryps ++++ | T. vivax |
| 6 | 39.6°C | 30.0% | Tryps ++++ | T. vivax |
| 7 | 39.7°C | 30.0% | Tryps +++ | T. vivax |
| 8 | 39.7°C | 30.0% | Tryps ++++ | T. vivax |
| 9 | 39.2°C | 27.5% | Tryps ++ | T. vivax |
| 10 | 39.9°C | 27.0% | Tryps +++ | T. vivax |
| 11 | 39.8°C | 26.0% | Tryps +++ | T. vivax |
| 12 | 39.5°C | 26.5% | Tryps + | T. vivax |
| 13 | 39.0°C | 26.0% | Tryps +++ | T. vivax |
| 14 | 39.5°C | 27.0% | Tryps ++ | T. vivax |
| 15 | 39.4°C | 26.5% | Tryps ++ | T. vivax |
| 16 | 39.9°C | 26.0% | Tryps ++ | T. vivax |
| 17 | 39.5°C | 27.0% | Tryps + | T. vivax |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 39.9°C | 26.0% | Tryps ++ | T. vivax |
| 21 | 39.8°C | 25.0% | Tryps ++ | T. vivax |
| 25 | 39.6°C | 24.0% | Tryps +++ | T. vivax |
| 28 | 39.2°C | 23.5% | Tryps ++ | T. vivax |
| 31 | 39.3°C | 22.5% | NPF | NPF |
| 36 | 38.3°C | 22.5% | NPF | NPF |
| 39 | 38.5°C | 34.0% | NPF | NPF |
| 42 | 38.4°C | 22.0% | NPF | NPF |
| 44 | 38.4°C | 21.0% | Tryps + | NPF |
| 49 | 38.4°C | 14.0% | NPF | NPF |
| 51 | 38.8°C | 14.0% | Tryps +++ | T. vivax |
| 53 | 38.6°C | 13.0% | Tryps +++ | T. vivax |
| 58 | 38.6°C | 13.0% | Tryps ++ | T. vivax |
| 60 | 38.2°C | 12.5% | Tryps ++ | T. vivax |
| 63 | 38.2°C | 12.0% | Tryps ++ | T. vivax |
| 65 | 38.0°C | 12.0% | Tryps + | T. vivax |
| 68 | 37.0°C | 12.0% | Tryps + | T. vivax |
| 70 | 38.9°C | 13.0% | Tryps + | T. vivax |
| 72 | 38.5°C | 13.0% | Tryps ++ | T. vivax |
| 77 | 38.6°C | 13.0% | Tryps + | T. vivax |
| 79 | 38.4°C | 12.0% | Tryps ++ | T. vivax |
| 81 | 38.6°C | 12.5% | Tryps +++ | T. vivax |
| 88 | 38.5°C | 11.5% | Tryps + | T. vivax |
| 91 | 38.2°C | 11.5% | Tryps + | T. vivax |
| 93 | 38.2°C | 11.0% | Tryps + | T. vivax |
| 95 | 38.0°C | 11.0% | Tryps + | T. vivax |
| 98 | 37.0°C | 11.0% | Tryps + | T. vivax |

HEIFER NO. 1671 (Control Heifer)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.5°C | 35.0% | NPF | T. mutans few |
| - 29 | 38.4°C | 32.0% | NPF | T. mutans few |
| - 22 | 38.5°C | 30.0% | NPF | NPF |
| - 15 | 38.5°C | 30.0% | NPF | NPF |
| - 8 | 38.7°C | 30.0% | NPF | NPF |
| - 3 | 38.6°C | 34.0% | NPF | NPF |
| 0 | 38.6°C | 34.0% | NPF | NPF |
| 1 | 38.7°C | 34.5% | NPF | NPF |
| 2 | 38.5°C | 31.5% | NPF | NPF |
| 3 | 38.3°C | 31.5% | NPF | NPF |
| 4 | 38.5°C | 33.0% | NPF | NPF |
| 5 | 38.5°C | 34.0% | NPF | NPF |
| 6 | 38.5°C | 32.0% | NPF | NPF |
| 7 | 38.4°C | 33.0% | NPF | NPF |
| 8 | 38.5°C | 33.0% | NPF | NPF |
| 9 | 38.5°C | 33.0% | NPF | NPF |
| 10 | 38.5°C | 33.5% | NPF | NPF |
| 11 | 38.4°C | 33.0% | NPF | NPF |
| 12 | 38.3°C | 35.0% | NPF | NPF |
| 13 | 38.3°C | 34.5% | NPF | NPF |
| 14 | 38.4°C | 34.5% | NPF | NPF |
| 15 | 38.4°C | 34.5% | NPF | NPF |
| 16 | 38.4°C | 34.5% | NPF | NPF |
| 17 | 38.6°C | 34.5% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 38.7°C | 31.0% | NPF | NPF |
| 21 | 38.5°C | 30.0% | NPF | NPF |
| 25 | 38.5°C | 20.0% | NPF | NPF |
| 28 | 38.5°C | 30.0% | NPF | NPF |
| 31 | 38.5°C | 29.0% | NPF | NPF |
| 36 | 38.3°C | 29.5% | NPF | NPF |
| 39 | 38.2°C | 29.5% | NPF | NPF |
| 42 | 38.0°C | 30.0% | NPF | NPF |
| 44 | 38.3°C | 27.0% | NPF | NPF |
| 49 | 38.3°C | 26.0% | NPF | NPF |
| 51 | 38.4°C | 26.0% | NPF | NPF |
| 53 | 38.2°C | 28.5% | NPF | NPF |
| 58 | 38.5°C | 28.5% | NPF | NPF |
| 60 | 38.3°C | 28.5% | NPF | NPF |
| 63 | 38.4°C | 30.0% | NPF | NPF |
| 65 | 38.4°C | 32.0% | NPF | NPF |
| 70 | 38.6°C | 30.0% | NPF | NPF |
| 72 | 38.4°C | 32.5% | NPF | NPF |
| 77 | 38.3°C | 33.0% | NPF | NPF |
| 79 | 38.3°C | 33.0% | NPF | NPF |
| 81 | 38.3°C | 30.0% | NPF | NPF |
| 85 | 38.3°C | 30.0% | NPF | NPF |
| 86 | 38.4°C | 30.5% | NPF | NPF |
| 88 | 38.4°C | 30.0% | NPF | NPF |
| 91 | 38.4°C | 31.0% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.5°C | 29.5% | NPF | NPF |
| 95 | 38.5°C | 30.0% | NPF | NPF |
| 98 | 38.4°C | 29.0% | NPF | NPF |
| 100 | 38.3°C | 30.0% | NPF | NPF |
| 102 | 38.3°C | 30.0% | NPF | NPF |
| 109 | 38.2°C | 30.0% | NPF | NPF |
| 115 | 38.5°C | 30.0% | NPF | NPF |
| 122 | 38.4°C | 30.5% | NPF | NPF |
| 129 | 38.5°C | 30.5% | NPF | NPF |
| 135 | 38.5°C | 31.0% | NPF | NPF |
| 157 | 38.5°C | 31.5% | NPF | NPF |
| 164 | 38.6°C | 33.0% | NPF | NPF |
| 171 | 38.7°C | 33.0% | NPF | NPF |

HEIFER NO. 1674 (Control Heifer)

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| - 35 | 38.6°C | 30.0% | NPF | T. mutans few |
| - 38. | 38.5°C | 30.5% | NPF | T. mutans few |
| - 22 | 38.4°C | 30.0% | NPF | T. mutans few |
| - 15 | 38.7°C | 30.0% | NPF | NPF |
| - 8 | 38.6°C | 30.0% | NPF | NPF |
| - 3 | 38.5°C | 30.0% | NPF | NPF |
| 0 | 38.5°C | 30.0% | NPF | NPF |
| 1 | 38.5°C | 30.0% | NPF | NPF |
| 2 | 38.3°C | 30.5% | NPF | NPF |
| 3 | 38.3°C | 31.0% | NPF | NPF |
| 4 | 38.7°C | 30.5% | NPF | NPF |
| 5 | 38.6°C | 31.5% | NPF | NPF |
| 6 | 38.7°C | 32.0% | NPF | NPF |
| 7 | 38.7°C | 29.5% | NPF | NPF |
| 8 | 38.3°C | 29.5% | NPF | NPF |
| 9 | 38.0°C | 30.0% | NPF | NPF |
| 10 | 38.2°C | 30.0% | NPF | NPF |
| 11 | 38.3°C | 29.5% | NPF | NPF |
| 12 | 38.3°C | 29.5% | NPF | NPF |
| 13 | 38.4°C | 29.5% | NPF | NPF |
| 14 | 38.7°C | 29.5% | NPF | NPF |
| 15 | 38.5°C | 29.0% | NPF | NPF |
| 16 | 38.2°C | 29.0% | NPF | NPF |
| 17 | 38.3°C | 27.0% | NPF | T. mutans few |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 18 | 38.4°C | 27.0% | NPF | NPF |
| 21 | 38.5°C | 25.0% | NPF | NPF |
| 25 | 38.6°C | 29.0% | NPF | NPF |
| 28 | 38.6°C | 30.0% | NPF | NPF |
| 31 | 38.6°C | 27.0% | NPF | NPF |
| 36 | 38.6°C | 27.0% | NPF | NPF |
| 39 | 38.3°C | 27.5% | NPF | T. mutans few |
| 42 | 38.4°C | 26.0% | NPF | T. mutans few |
| 44 | 38.4°C | 30.0% | NPF | T. mutans few |
| 49 | 38.4°C | 31.0% | NPF | T. mutans few |
| 51 | 38.4°C | 27.0% | NPF | T. mutans few |
| 53 | 38.5°C | 27.0% | NPF | T. mutans few |
| 58 | 38.6°C | 27.0% | NPF | T. mutans few |
| 60 | 38.5°C | 27.0% | NPF | T. mutans few |
| 63 | 38.5°C | 27.0% | NPF | T. mutans few |
| 65 | 38.4°C | 28.0% | NPF | T. mutans few |
| 70 | 38.5°C | 28.0% | NPF | T. mutans few |
| 72 | 38.5°C | 28.5% | NPF | T. mutans few |
| 77 | 38.5°C | 28.5% | NPF | T. mutans few |
| 79 | 38.5°C | 28.5% | NPF | T. mutans few |
| 81 | 38.5°C | 27.0% | NPF | T. mutans few |
| 85 | 38.5°C | 26.5% | NPF | T. mutans few |
| 86 | 38.3°C | 28.0% | NPF | T. mutans few |
| 88 | 38.4°C | 28.0% | NPF | T. mutans few |
| 91 | 38.2°C | 28.5% | NPF | NPF |

| Day | Temperature | %PCV | HCT | Thin Smear |
|---|---|---|---|---|
| 93 | 38.3°C | 29.0% | NPF | NPF |
| 95 | 38.4°C | 29.0% | NPF | NPF |
| 98 | 38.4°C | 29.0% | NPF | NPF |
| 100 | 38.5°C | 27.5% | NPF | NPF |
| 102 | 38.7°C | 26.6% | NPF | NPF |
| 109 | 38.6°C | 27.0% | NPF | NPF |
| 115 | 38.7°C | 28.0% | NPF | NPF |
| 122 | 38.7°C | 28.0% | NPF | NPF |
| 129 | 38.5°C | 27.5% | NPF | NPF |
| 135 | 38.5°C | 27.5% | NPF | NPF |
| 157 | 38.4°C | 28.0% | NPF | NPF |
| 164 | 38.5°C | 28.0% | NPF | NPF |
| 171 | 38.7°C | 28.0% | NPF | NPF |

# APPENDIX 5

## IgM AND IgG LEVELS IN NONPREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH TRYPANOSOMA VIVAX CLOSE TO THE TIME OF ESTRUS AND THOSE OF THE CONTROLS

IgM LEVELS IN HEIFER NO. L31

| Week | Ring Diameter (mm) | Concentration mg/100 | Final Conc. mg/100ml | Δ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | 62.5 |
| -1 | 5.0 | 60 | 300 | 0.0 |
| 0 | 5.5 | 60 | 300 | 62.5 |
| 1 | 5.5 | 85 | 425 | 125.0 |
| 2 | 6.0 | 125 | 625 | 225.0 |
| 3 | 6.5 | 185 | 925 | 459.5 |
| 4 | 6.5 | 185 | 925 | 625.0 |
| 5 | 7.5 | 370 | 1,850 | 1,550.0 |
| 6 | 7.5 | 370 | 1,850 | 1,550.0 |
| 7 | 7.5 | 370 | 1,850 | 1,550.0 |
| 8 | 7.5 | 370 | 1,850 | 1,487.5 |
| 9 | 7.5 | 370 | 1,850 | 1,425.0 |
| 10 | 7.5 | 370 | 1,850 | 1,550.0 |
| 11 | 7.5 | 370 | 1,850 | 1,550.0 |
| 12 | 7.5 | 370 | 1,850 | 1,550.0 |
| 13 | 7.0 | 260 | 1,300 | 1,000.0 |
| 14 | 7.0 | 260 | 1,300 | 1,000.0 |
| 15 | 7.0 | 260 | 1,300 | 1,000.0 |

IgM LEVELS IN HEIFER NO. L25

| We k | Ring Diameter (mm) | Concentration mg/100 | Final Conc. mg/100ml | Δ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | 62.5 |
| -1 | 5.0 | 60 | 300 | 0.0 |
| 0 | 5.0 | 60 | 300 | 62.5 |
| 1 | 7.0 | 260 | 1,300 | 1,000.0 |
| 2 | 7.5 | 370 | 1,850 | 1,550.0 |
| 3 | 8.0 | 520 | 2,600 | 2,137.5 |
| 4 | 8.0 | 520 | 2,600 | 2,300.0 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,237.5 |
| 8 | 8.0 | 520 | 2,600 | 2,174.5 |
| 9 | 8.0 | 520 | 2,600 | 2,300.0 |
| 10 | 8.0 | 520 | 2,600 | 2,300.0 |
| 11 | 7.5 | 370 | 1,850 | 1,550.0 |
| 12 | 7.5 | 370 | 1,850 | 1,550.0 |
| 13 | 7.5 | 370 | 1,850 | 1,550.0 |
| 14 | 7.5 | 370 | 1,850 | 1,550.0 |
| 15 | 7.0 | 260 | 1,300 | 1,000.0 |

IgM LEVELS IN HEIFER NO. L20

| Week | Ring Diameter | Concentration mg/100 | Final Conc. mg/100ml | Δ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | 62.5 |
| +1 | 5.0 | 60 | 300 | 0.0 |
| 0 | 5.0 | 60 | 300 | 62.0 |
| 1 | 7.0 | 260 | 1,300 | 1,000.0 |
| 2 | 7.5 | 370 | 1,850 | 1,550.0 |
| 3 | 8.0 | 520 | 2,600 | 2,137.5 |
| 4 | 8.0 | 520 | 2,600 | 2,300.0 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,237.5 |
| 8 | 8.0 | 520 | 2,600 | 2,175.0 |
| 9 | 8.0 | 520 | 2,600 | 2,300.0 |
| 10 | 8.0 | 520 | 2,600 | 2,300.0 |
| 11 | 7.5 | 370 | 1,850 | 1,550.0 |
| 12 | 7.5 | 370 | 1,850 | 1,550.0 |
| 13 | 7.5 | 370 | 1,850 | 1,550.0 |
| 14 | 7.0 | 260 | 1,300 | 1,000.0 |
| 15 | 7.0 | 260 | 1,300 | 1,000.0 |

IgM LEVELS IN HEIFER NO. L32

| Week | Ring Diameter (mm) | Concentration mg/100 | Final Conc. mg/100ml | △ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | 62.5 |
| -1 | 5.0 | 60 | 300 | 0.0 |
| 0 | 5.0 | 60 | 300 | 62.5 |
| 1 | 5.0 | 60 | 425 | 125.0 |
| 2 | 7.5 | 370 | 1,850 | 1,550.0 |
| 3 | 8.0 | 520 | 2,600 | 2,137.5 |
| 4 | 8.0 | 520 | 2,600 | 2,300.0 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,237.0 |
| 8 | 8.0 | 520 | 2,600 | 2,175.0 |
| 9 | 8.0 | 520 | 2,600 | 2,300.0 |
| 10 | 7.5 | 370 | 1,850 | 1,550.0 |
| 11 | 8.0 | 520 | 2,500 | 2,300.0 |
| 12 | 7.5 | 370 | 1,850 | 1,550.0 |
| 13 | 7.5 | 370 | 1,850 | 1,550.0 |
| 14 | 6.0 | 185 | 925 | 625.0 |
| 15 | 6.0 | 185 | 925 | 625.0 |

IgM LEVELS IN HEIFER NO. L15

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 5.0 | 60 | 300 |
| -1 | 5.0 | 60 | 300 |
| 0 | 5.5 | 85 | 425 |
| 1 | 5.0 | 60 | 300 |
| 2 | 5.0 | 60 | 300 |
| 3 | 5.0 | 60 | 300 |
| 4 | 5.0 | 60 | 300 |
| 5 | 5.0 | 60 | 300 |
| 6 | 5.0 | 60 | 300 |
| 7 | 5.5 | 85 | 425 |
| 8 | 5.5 | 85 | 425 |
| 9 | 5.0 | 60 | 300 |
| 10 | 5.0 | 60 | 300 |
| 11 | 5.0 | 60 | 300 |
| 12 | 5.0 | 60 | 300 |
| 13 | 5.0 | 60 | 300 |
| 14 | 5.0 | 60 | 300 |
| 15 | 5.0 | 60 | 300 |

IgM LEVELS IN HEIFER NO. L66

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 5.5 | 85 | 425 |
| -1 | 5.0 | 60 | 300 |
| 0 | 5.0 | 60 | 300 |
| 1 | 5.0 | 60 | 300 |
| 2 | 5.0 | 60 | 300 |
| 3 | 5.0 | 60 | 300 |
| 4 | 5.0 | 60 | 300 |
| 5 | 5.0 | 60 | 300 |
| 6 | 5.0 | 60 | 300 |
| 7 | 5.0 | 60 | 300 |
| 8 | 5.0 | 60 | 300 |
| 9 | 5.0 | 60 | 300 |
| 10 | 5.0 | 60 | 300 |
| 11 | 5.0 | 60 | 300 |
| 12 | 5.0 | 60 | 300 |
| 13 | 5.0 | 60 | 300 |
| 14 | 5.0 | 60 | 300 |
| 15 | 5.0 | 60 | 300 |

IgG LEVELS IN HEIFER NO. L31

| Week | Ring Diameter (mm) | Concentration mg/100ml | Final Conc. mg/100ml | △IgG |
|---|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 | 275.0 |
| -1 | 6.0 | 500 | 2,500 | 0.0 |
| 0 | 6.0 | 500 | 2,500 | 0.0 |
| 1 | 6.5 | 610 | 3,050 | 550.0 |
| 2 | 6.5 | 610 | 3,050 | 550.0 |
| 3 | 6.5 | 610 | 3,050 | 550.0 |
| 4 | 7.0 | 750 | 3,750 | 975.0 |
| 5 | 7.5 | 920 | 4,600 | 2,100.0 |
| 6 | 7.5 | 920 | 4,600 | 2,100.0 |
| 7 | 7.5 | 920 | 4,600 | 2,100.0 |
| 8 | 7.5 | 920 | 4,600 | 2,100.0 |
| 9 | 7.5 | 920 | 4,600 | 2,100.0 |
| 10 | 7.5 | 920 | 4,600 | 2,100.0 |
| 11 | 7.5 | 920 | 4,600 | 2,100.0 |
| 12 | 7.5 | 920 | 4,600 | 2,100.0 |
| 13 | 7.0 | 750 | 3,750 | 1,250.0 |
| 14 | 7.0 | 750 | 3,750 | 1,250.0 |
| 15 | 7.0 | 750 | 3,750 | 975.0 |

IgG LEVELS IN HEIFER NO. L25

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. mg/100ml | Δ IgG |
|---|---|---|---|---|
| -2 | 6.5 | 610 | 3,050 | 275.0 |
| +1 | 6.5 | 610 | 3,050 | 550.0 |
| 0 | 6.5 | 610 | 3,050 | 550.0 |
| 1 | 6.5 | 610 | 3,050 | 550.0 |
| 2 | 7.0 | 750 | 3,750 | 1,250.0 |
| 3 | 7.0 | 750 | 3,750 | 1,250.0 |
| 4 | 7.5 | 920 | 4,600 | 1,825.0 |
| 5 | 7.0 | 750 | 3,750 | 1,250.0 |
| 6 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 7 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 8 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 10 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 11 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 12 | 7.5 | 920 | 4,600 | 2,100.0 |
| 13 | 7.5 | 920 | 4,600 | 2,100.0 |
| 14 | 7.0 | 750 | 3,750 | 1,250.0 |
| 15 | 7.0 | 750 | 3,750 | 975.0 |

IgG LEVELS IN HEIFER NO. L2C

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgG |
|---|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 | 275.0 |
| -1 | 6.0 | 500 | 2,500 | 0.0 |
| 0 | 6.0 | 500 | 2,500 | 0.0 |
| 1 | 6.0 | 500 | 2,500 | 0.0 |
| 2 | 6.5 | 610 | 3,050 | 550.0 |
| 3 | 6.5 | 610 | 3,050 | 550.0 |
| 4 | 7.0 | 750 | 3,750 | 975.0 |
| 5 | 7.5 | 920 | 4,600 | 2,100.0 |
| 6 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 7 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 8 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 10 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 11 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 12 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 13 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 14 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 15 | 8.0 | 1100 | 5,500 | 2,725.0 |

IgG LEVELS IN HEIFER NO. L32

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgG |
|---|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 | 275.0 |
| -1 | 6.0 | 500 | 2,500 | 0.0 |
| 0 | 6.0 | 500 | 2,500 | 0.0 |
| 1 | 6.0 | 500 | 2,500 | 0.0 |
| 2 | 7.0 | 750 | 3,750 | 1,250.0 |
| 3 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 4 | 7.5 | 920 | 4,600 | 1,825.0 |
| 5 | 7.0 | 750 | 3,750 | 1,250.0 |
| 6 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 7 | 8.5 | 1400 | 7,000 | 4,500.0 |
| 8 | 8.5 | 1400 | 7,000 | 4,500.0 |
| 9 | 7.0 | 750 | 3,750 | 975.0 |
| 10 | 7.0 | 750 | 3,750 | 1,250.0 |
| 11 | 7.0 | 750 | 3,750 | 1,250.0 |
| 12 | 6.5 | 610 | 3,050 | 550.0 |
| 13 | 6.5 | 610 | 3,050 | 550.0 |
| 14 | 6.5 | 610 | 3,050 | 550.0 |
| 15 | 6.5 | 610 | 3,050 | 275.0 |

IgG LEVELS IN HEIFER NO. L15

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100 ml) |
|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 |
| -1 | 6.0 | 500 | 2,500 |
| 0 | 6.0 | 500 | 2,500 |
| 1 | 6.0 | 500 | 2,500 |
| 2 | 6.0 | 500 | 2,500 |
| 3 | 6.0 | 500 | 2,500 |
| 4 | 6.0 | 500 | 2,500 |
| 5 | 6.0 | 500 | 2,500 |
| 6 | 6.0 | 500 | 2,500 |
| 7 | 6.0 | 500 | 2,500 |
| 8 | 6.0 | 500 | 2,500 |
| 9 | 6.0 | 500 | 2,500 |
| 10 | 6.0 | 500 | 2,500 |
| 11 | 6.0 | 500 | 2,500 |
| 12 | 6.0 | 500 | 2,500 |
| 13 | 6.0 | 500 | 2,500 |
| 14 | 6.0 | 500 | 2,500 |
| 15 | 6.0 | 500 | 2,500 |

IgG LEVELS IN HEIFER NO. L66

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100 ml) |
|---|---|---|---|
| -2 | 6.5 | 610 | 3,050 |
| -1 | 6.0 | 500 | 2,500 |
| 0 | 6.0 | 500 | 2,500 |
| 1 | 6.0 | 500 | 2,500 |
| 2 | 6.0 | 500 | 2,500 |
| 3 | 6.0 | 500 | 2,500 |
| 4 | 6.5 | 610 | 3,050 |
| 5 | 6.0 | 500 | 2,500 |
| 6 | 6.0 | 500 | 2,500 |
| 7 | 6.0 | 500 | 2,500 |
| 8 | 6.0 | 500 | 2,500 |
| 9 | 6.5 | 610 | 3,050 |
| 10 | 6.0 | 500 | 2,500 |
| 11 | 6.0 | 500 | 2,500 |
| 12 | 6.0 | 500 | 2,500 |
| 13 | 6.0 | 500 | 2,500 |
| 14 | 6.0 | 500 | 2,500 |
| 15 | 6.5 | 610 | 3,050 |

# APPENDIX 6

IgM AND IgG LEVELS IN PREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH TRYPANOSOMA VIVAX DURING THE FIRST TRIMESTER AND THOSE OF THE CONTROLS

IgM LEVELS IN HEIFER NO. 1933

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | 0.0 |
| -1 | 5.0 | 60 | 300 | 0.0 |
| 0 | 5.0 | 60 | 300 | 0.0 |
| 1 | 5.5 | 85 | 425 | 62.5 |
| 2 | 6.0 | 125 | 625 | 325.0 |
| 3 | 6.5 | 185 | 925 | 625.0 |
| 4 | 7.0 | 260 | 1,300 | 1,000.0 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,300.0 |
| 8 | 8.0 | 520 | 2,600 | 2,300.0 |
| 9 | 8.0 | 520 | 2,600 | 2,300.0 |
| 10 | 8.0 | 520 | 2,600 | 2,300.0 |
| 11 | 8.0 | 520 | 2,600 | 2,300.0 |
| 12 | 8.0 | 520 | 2,600 | 2,300.0 |
| 13 | 8.0 | 520 | 2,600 | 2,300.0 |
| 14 | 8.0 | 520 | 2,600 | 2,300.0 |
| 15 | 8.0 | 520 | 2,600 | 2,300.0 |

IgM LEVELS IN HEIFER NO. 1714

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | 0.0 |
| -1 | 5.0 | 60 | 300 | 0.0 |
| 0 | 5.0 | 60 | 300 | 0.0 |
| 1 | 6.0 | 125 | 625 | 325.0 |
| 2 | 8.0 | 520 | 2,600 | 2,300.0 |
| 3 | 8.0 | 520 | 2,500 | 2,300.0 |
| 4 | 8.0 | 520 | 2,600 | 2,300.0 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,300.0 |
| 8 | 8.0 | 520 | 2,600 | 2,300.0 |
| 9 | 8.0 | 520 | 2,600 | 2,300.0 |
| 10 | 8.0 | 520 | 2,600 | 2,300.0 |
| 11 | 8.0 | 520 | 2,600 | 2,300.0 |
| 12 | 7.0 | 260 | 1,300 | 1,000.0 |
| 13 | 7.5 | 370 | 1,850 | 1,550.0 |
| 14 | 8.0 | 520 | 2,600 | 2,300.0 |
| 15 | 8.0 | 520 | 2,600 | 2,300.0 |

IgM LEVELS IN HEIFER NO. 1817

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | 0.0 |
| -1 | 5.0 | 60 | 300 | 0.0 |
| 0 | 5.0 | 60 | 300 | 0.0 |
| 1 | 7.0 | 260 | 1,300 | 1,000.0 |
| 2 | 7.0 | 260 | 1,300 | 1,000.0 |
| 3 | 8.0 | 520 | 2,600 | 2,300.0 |
| 4 | 7.0 | 260 | 1,300 | 1,000.0 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,300.0 |
| 8 | 8.0 | 520 | 2,600 | 2,300.0 |
| 9 | 8.0 | 520 | 2,600 | 2,300.0 |
| 10 | 8.0 | 520 | 2,600 | 2,300.0 |
| 11 | 8.0 | 520 | 2,600 | 2,300.0 |
| 12 | 8.0 | 520 | 2,600 | 2,300.0 |
| 13 | 7.0 | 260 | 1,300 | 1,000.0 |
| 14 | 7.5 | 370 | 1,850 | 1,550.0 |
| 15 | 7.5 | 370 | 1,850 | 1,550.0 |

IgM LEVELS IN HEIFER NO. 1703

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | ∠IgM |
|---|---|---|---|---|
| -2 | 5.5 | 85 | 425 | 125.0 |
| -1 | 5.5 | 85 | 425 | 125.0 |
| 0 | 5.5 | 85 | 425 | 125.0 |
| 1 | 7.0 | 260 | 1,300 | 1,000.0 |
| 2 | 8.0 | 520 | 2,600 | 2,300.0 |
| 3 | 8.0 | 520 | 2,600 | 2,300.0 |
| 4 | 8.0 | 520 | 2,600 | 2,300.0 |
| 5 | 7.0 | 260 | 1,300 | 1,000.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,300.0 |
| 8 | 8.0 | 520 | 2,600 | 2,300.0 |
| 9 | 8.0 | 520 | 2,600 | 2,300.0 |
| 10 | 8.0 | 520 | 2,600 | 2,300.0 |
| 11 | 8.0 | 520 | 2,600 | 2,300.0 |
| 12 | 8.0 | 520 | 2,600 | 2,300.0 |
| 13 | 8.0 | 520 | 2,600 | 2,300.0 |
| 14 | 8.0 | 520 | 2,600 | 2,300.0 |
| 15 | 8.0 | 520 | 2,600 | 2,300.0 |

IgM LEVELS IN HEIFER NO. 1801

(Control Heifer)

| Week | Ringo Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 5.0 | 60 | 300 |
| +1 | 5.0 | 60 | 300 |
| 0 | 5.0 | 60 | 300 |
| 1 | 5.0 | 60 | 300 |
| 2 | 5.0 | 60 | 300 |
| 3 | 5.0 | 60 | 300 |
| 4 | 5.0 | 60 | 300 |
| 5 | 5.0 | 60 | 300 |
| 6 | 5.0 | 60 | 300 |
| 7 | 5.0 | 60 | 300 |
| 8 | 5.0 | 60 | 300 |
| 9 | 5.0 | 60 | 300 |
| 10 | 5.0 | 60 | 300 |
| 11 | 5.0 | 60 | 300 |
| 12 | 5.0 | 60 | 300 |
| 13 | 5.0 | 60 | 300 |
| 14 | 5.0 | 60 | 300 |
| 15 | 5.0 | 60 | 300 |

IgM LEVELS IN HEIFER NO. 1764

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 5.0 | 60 | 300 |
| +1 | 5.0 | 60 | 300 |
| 0 | 5.0 | 60 | 300 |
| 1 | 5.5 | 85 | 425 |
| 2 | 5.0 | 60 | 300 |
| 3 | 5.0 | 60 | 300 |
| 4 | 5.0 | 60 | 300 |
| 5 | 5.0 | 60 | 300 |
| 6 | 5.0 | 60 | 300 |
| 7 | 5.0 | 60 | 300 |
| 8 | 5.0 | 60 | 300 |
| 9 | 5.0 | 60 | 300 |
| 10 | 5.0 | 60 | 300 |
| 11 | 5.0 | 60 | 300 |
| 12 | 5.0 | 60 | 300 |
| 13 | 5.0 | 60 | 300 |
| 14 | 5.0 | 60 | 300 |
| 15 | 5.0 | 60 | 300 |

IgG LEVELS IN HEIFER NO. 1933

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgG |
|---|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 | -900.0 |
| -1 | 6.0 | 500 | 2,500 | -900.0 |
| 0 | 6.0 | 500 | 2,500 | -900.0 |
| 1 | 6.0 | 500 | 2,500 | -900.0 |
| 2 | 6.5 | 610 | 3,050 | -350.0 |
| 3 | 6.5 | 610 | 3,050 | -350.0 |
| 4 | 7.0 | 750 | 3,750 | 350.0 |
| 5 | 7.0 | 750 | 3,750 | 350.0 |
| 6 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 7 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 8 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,375.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,375.00 |
| 11 | 8.0 | 1100 | 5,500 | 2,100.00 |
| 12 | 8.0 | 1100 | 5,500 | 2,100.00 |
| 13 | 7.5 | 920 | 4,600 | 1,200.0 |
| 14 | 8.0 | 1100 | 5,500 | 1,750.0 |
| 15 | 7.5 | 920 | 4,600 | 850.0 |

IgG LEVELS FOR HEIFER NO. 1714

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | /\ IgG |
|---|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 | -900.0 |
| -1 | 6.0 | 500 | 2,500 | -900.0 |
| 0 | 6.0 | 500 | 2,500 | -900.0 |
| 1 | 6.5 | 610 | 3,050 | -350.0 |
| 2 | 7.5 | 920 | 4,600 | 1,200.0 |
| 3 | 7.5 | 920 | 4,600 | 1,200.0 |
| 4 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 5 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 6 | 8.0 | 1100 | 5,500 | 4,250.0 |
| 7 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 8 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,375.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,375.0 |
| 11 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 12 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 13 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 14 | 8.0 | 1100 | 5,500 | 1,750.0 |
| 15 | 8.0 | 1100 | 5,500 | 1,750.0 |

IgG LEVELS IN HEIFER NO. 1817

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgG |
|---|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 | 350.0 |
| -1 | 6.5 | 610 | 3,050 | -350.0 |
| 0 | 7.0 | 750 | 3,750 | 350.0 |
| 1 | 7.0 | 750 | 3,750 | 350.0 |
| 2 | 7.5 | 920 | 4,600 | 1,200.0 |
| 3 | 7.0 | 750 | 3,750 | 350.0 |
| 4 | 7.5 | 920 | 4,600 | 1,200.0 |
| 5 | 7.5 | 920 | 4,600 | 1,200.0 |
| 6 | 8.5 | 1400 | 7,000 | 3,950.0 |
| 7 | 8.5 | 1400 | 7,000 | 3,600.0 |
| 8 | 8.5 | 1400 | 7,000 | 3,600.0 |
| 9 | 8.5 | 1400 | 7,000 | 3,875.0 |
| 10 | 8.5 | 1400 | 7,000 | 3,875.0 |
| 11 | 8.5 | 1400 | 7,000 | 3,600.0 |
| 12 | 8.5 | 1400 | 7,000 | 3,600.0 |
| 13 | 8.5 | 1400 | 7,000 | 3,600.0 |
| 14 | 8.5 | 1400 | 7,000 | 3,250.0 |
| 15 | 8.5 | 1400 | 7,000 | 3,250.0 |

IgG LEVELS FOR HEIFER NO. 1703

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgG |
|---|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 | 350.0 |
| -1 | 7.0 | 750 | 3,750 | 350.0 |
| 0 | 7.0 | 750 | 3,750 | 350.0 |
| 1 | 7.5 | 920 | 4,600 | 1,200.0 |
| 2 | 7.5 | 920 | 4,600 | 1,200.0 |
| 3 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 4 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 5 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 6 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 7 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 8 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,375.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,375.0 |
| 11 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 12 | 7.0 | 750 | 3,750 | 350.0 |
| 13 | 7.0 | 750. | 3,750 | 350.0 |
| 14 | 7.5 | 920 | 4,600 | 850.0 |
| 15 | 7.5 | 920 | 4,600 | 850.0 |

## IgG LEVELS IN HEIFER NO. 1801

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 6.5 | 610 | 3,050 |
| -1 | 6.5 | 610 | 3,050 |
| 0 | 6.5 | 610 | 3,050 |
| 1 | 6.5 | 610 | 3,050 |
| 2 | 6.5 | 610 | 3,050 |
| 3 | 6.5 | 610 | 3,050 |
| 4 | 6.5 | 610 | 3,050 |
| 5 | 6.5 | 610 | 3,050 |
| 6 | 6.5 | 610 | 3,050 |
| 7 | 6.5 | 610 | 3,050 |
| 8 | 6.5 | 610 | 3,050 |
| 9 | 6.0 | 500 | 2,500 |
| 10 | 6.0 | 500 | 2,500 |
| 11 | 7.0 | 750 | 3,750 |
| 12 | 7.0 | 750 | 3,750 |
| 13 | 7.0 | 750 | 3,750 |
| 14 | 7.0 | 750 | 3,750 |
| 15 | 7.0 | 750 | 3,750 |

IgG LEVELS IN HEIFER NO. 1764

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 |
| -1 | 7.0 | 750 | 3,750 |
| 0 | 7.0 | 750 | 3,750 |
| 1 | 7.0 | 750 | 3,750 |
| 2 | 7.0 | 750 | 3,750 |
| 3 | 7.0 | 750 | 3,750 |
| 4 | 7.0 | 750 | 3,750 |
| 5 | 7.0 | 750 | 3,750 |
| 6 | 6.5 | 610 | 3,050 |
| 7 | 7.0 | 750 | 3,750 |
| 8 | 7.0 | 750 | 3,750 |
| 9 | 7.0 | 750 | 3,750 |
| 10 | 7.0 | 750 | 3,750 |
| 11 | 6.5 | 610 | 3,050 |
| 12 | 6.5 | 610 | 3,050 |
| 13 | 6.5 | 610 | 3,050 |
| 14 | 7.0 | 750 | 3,750 |
| 15 | 7.0 | 750 | 3,750 |

# APPENDIX 7

IgM AND IgG LEVELS IN PREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH TRYPANOSOMA VIVAX DURING THE SECOND TRIMESTER AND THOSE OF THE CONTROLS

IgM LEVELS IN HEIFER NO. 318

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgM |
|---|---|---|---|---|
| -2 | 5.5 | 85 | 425 | 62.5 |
| -1 | 5.0 | 60 | 300 | -125.0 |
| 0 | 5.0 | 60 | 300 | 0.0 |
| 1 | 5.0 | 60 | 300 | 62.0 |
| 2 | 7.5 | 370 | 1,850 | 1,487.5 |
| 3 | 8.0 | 520 | 2,600 | 2,237.5 |
| 4 | 8.0 | 520 | 2,600 | 2,237.5 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,237.5 |
| 8 | 8.0 | 520 | 2,600 | 2,237.5 |
| 9 | 8.0 | 520 | 2,600 | 2,237.5 |
| 10 | 8.0 | 520 | 2,600 | 2,237.5 |
| 11 | 8.0 | 520 | 2,600 | 2,237.5 |
| 12 | 8.0 | 520 | 2,600 | 2,175.0 |
| 13 | 8.0 | 520 | 2,600 | 2,175.0 |
| 14 | 8.0 | 520 | 2,600 | 2,175.0 |
| 15 | 7.0 | 260 | 1,300 | 875.0 |

## IgM LEVELS IN HEIFER NO. 327

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgM |
|---|---|---|---|---|
| -2 | 5.5 | 85 | 425 | 62.5 |
| -1 | 5.5 | 85 | 4 5 | 0.0 |
| 0 | 5.5 | 85 | 425 | 125.0 |
| 1 | 5.5 | 85 | 425 | 62.5 |
| 2 | 6.5 | 185 | 925 | 562.5 |
| 3 | 7.5 | 370 | 1,850 | 1,487.5 |
| 4 | 8.0 | 520 | 2,600 | 2,237.5 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,237.5 |
| 8 | 8.0 | 520 | 2,600 | 2,237.5 |
| 9 | 8.0 | 520 | 2,600 | 2,237.5 |
| 10 | 8.0 | 520 | 2,600 | 2,237.5 |
| 11 | 8.0 | 520 | 2,600 | 2,237.5 |
| 12 | 8.0 | 520 | 2,600 | 2,175.0 |
| 13 | 8.0 | 520 | 2,600 | 2,175.0 |
| 14 | 8.0 | 520 | 2,600 | 2,175.0 |
| 15 | 8.0 | 520 | 2,600 | 2,175.0 |

IgM LEVELS IN HEIFER NO. 337

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgM |
|---|---|---|---|---|
| -2 | 5.5 | 85 | 425 | 62.5 |
| -1 | 5.5 | 85 | 425 | 0.0 |
| 0 | 5.5 | 85 | 425 | 125.0 |
| 1 | 5.5 | 85 | 425 | 62.5 |
| 2 | 6.5 | 185 | 925 | 562.5 |
| 3 | 6.5 | 185 | 2,600 | 2,237.5 |
| 4 | 8.0 | 520 | 2,600 | 2,300.0 |
| 5 | 8.0 | 520 | 2,600 | 2,237.5 |
| 6 | 8.0 | 520 | 2,600 | 2,237.5 |
| 7 | 8.0 | 520 | 2,600 | 2,237.5 |
| 8 | 8.0 | 520 | 2,600 | 2,237.5 |
| 9 | 8.0 | 520 | 1,850 | 1,487.5 |
| 10 | 7.5 | 370 | 1,850 | 1,487.5 |
| 11 | 7.5 | 370 | 1,850 | 1,425.0 |
| 12 | 7.5 | 370 | 1,850 | 1,425.0 |
| 13 | 7.5 | 370 | 1,850 | 1,425.0 |
| 14 | 7.5 | 370 | 1,300 | 875.0 |
| 15 | 7.0 | 260 | | |

IgM LEVELS IN HEIFER NO. 325

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgM |
|---|---|---|---|---|
| -2 | 5.0 | 60 | 300 | -62.5 |
| -1 | 5.0 | 60 | 300 | -125.0 |
| 0 | 5.0 | 60 | 300 | 0.0 |
| 1 | 6.0 | 125 | 625 | 262.5 |
| 2 | 7.5 | 370 | 1,850 | 1,487.5 |
| 3 | 8.0 | 520 | 2,600 | 2,237.5 |
| 4 | 8.0 | 520 | 2,600 | 2,237.5 |
| 5 | 8.0 | 520 | 2,600 | 2,300.0 |
| 6 | 8.0 | 520 | 2,600 | 2,300.0 |
| 7 | 8.0 | 520 | 2,600 | 2,237.5 |
| 8 | 7.5 | 370 | 1,850 | 1,487.5 |
| 9 | 7.5 | 370 | 1,850 | 1,487.5 |
| 10 | 7.5 | 370 | 1,850 | 1,487.5 |
| 11 | 8.0 | 520 | 2,600 | 2,237.5 |
| 12 | 8.0 | 520 | 2,600 | 2,175.0 |
| 13 | 7.5 | 370 | 1,850 | 1,425.0 |
| 14 | 7.5 | 370 | 1,850 | 1,425.0 |
| 15 | 7.5 | 370 | 1,850 | 1,425.0 |

IgM LEVELS IN HEIFER NO. 139

(Control Animal)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 5.5 | 85 | 425 |
| -1 | 5.5 | 85 | 425 |
| 0 | 5.0 | 60 | 300 |
| 1 | 5.5 | 85 | 425 |
| 2 | 5.5 | 85 | 425 |
| 3 | 5.5 | 85 | 425 |
| 4 | 5.5 | 85 | 425 |
| 5 | 5.0 | 60 | 300 |
| 6 | 5.0 | 60 | 300 |
| 7 | 5.5 | 85 | 425 |
| 8 | 5.5 | 85 | 425 |
| 9 | 5.5 | 85 | 425 |
| 10 | 5.5 | 85 | 425 |
| 11 | 5.5 | 85 | 425 |
| 12 | 5.0 | 60 | 300 |
| 13 | 5.5 | 85 | 425 |
| 14 | 5.5 | 85 | 425 |
| 15 | 5.5 | 85 | 425 |

IgM LEVELS IN HEIFER NO. 227

(Control Animal)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 5.0 | 60 | 300 |
| -1 | 5.5 | 85 | 425 |
| 0 | 5.0 | 60 | 300 |
| 1 | 5.0 | 60 | 300 |
| 2 | 5.0 | 60 | 300 |
| 3 | 5.0 | 60 | 300 |
| 4 | 5.0 | 60 | 300 |
| 5 | 5.0 | 60 | 300 |
| 6 | 5.0 | 60 | 300 |
| 7 | 5.0 | 60 | 300 |
| 8 | 5.0 | 60 | 300 |
| 9 | 5.0 | 60 | 300 |
| 10 | 5.0 | 60 | 300 |
| 11 | 5.5 | 85 | 300 |
| 12 | 5.5 | 85 | 425 |
| 13 | 5.5 | 85 | 425 |
| 14 | 5.5 | 85 | 425 |
| 15 | 5.5 | 85 | 425 |

IgG LEVELS IN HEIFER NO. 318

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | *Final Conc. (mg/100ml) | △ IgG |
|---|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 | 0.0 |
| -1 | 6.5 | 610 | 3,050 | 550.0 |
| 0 | 6.0 | 500 | 2,500 | 0.0 |
| 1 | 7.5 | 920 | 4,600 | 2,100.0 |
| 2 | 7.5 | 920 | 4,600 | 2,100.0 |
| 3 | 7.5 | 920 | 4,600 | 2,100.0 |
| 4 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 5 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 6 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 7 | 7.5 | 920 | 4,600 | 2,100.0 |
| 8 | 7.5 | 920 | 4,600 | 2,100.0 |
| 9 | 7.5 | 920 | 4,600 | 1,825.0 |
| 10 | 7.5 | 920 | 4,600 | 1,825.0 |
| 11 | 7.5 | 920 | 4,600 | 1,825.0 |
| 12 | 7.5 | 920 | 4,600 | 1,825.0 |
| 13 | 7.5 | 920 | 4,600 | 1,825.0 |
| 14 | 6.5 | 610 | 3,050 | 275.0 |
| 15 | 6.5 | 610 | 3,050 | 275.0 |

IgG LEVELS IN HEIFER NO. 327

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgG |
|---|---|---|---|---|
| -2 | 6.5 | 610 | 3,050 | 550.0 |
| -1 | 6.0 | 500 | 2,500 | 0.0 |
| 0 | 6.0 | 500 | 2,500 | 0.0 |
| 1 | 6.0 | 500 | 2,500 | 0.0 |
| 2 | 7.5 | 920 | 4,600 | 2,100.0 |
| 3 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 4 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 5 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 6 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 7 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 8 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 11 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 12 | 7.5 | 920 | 4,600 | 1,825.0 |
| 13 | 7.0 | 750 | 3,750 | 975.0 |
| 14 | 6.5 | 610 | 3,050 | 275.0 |
| 15 | 6.5 | 610 | 3,050 | 275.0 |

IgG LEVELS IN HEIFER NO. 337

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgG |
|---|---|---|---|---|
| -2 | 6.5 | 610 | 3,050 | 550.0 |
| -1 | 6.5 | 610 | 3,050 | 550.0 |
| 0 | 6.5 | 610 | 3,050 | 550.0 |
| 1 | 6.5 | 610 | 3,050 | 550.0 |
| 2 | 7.5 | 920 | 4,600 | 2,100.0 |
| 3 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 4 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 5 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 6 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 7 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 8 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 10 | 7.5 | 920 | 4,600 | 1,825.0 |
| 11 | 7.5 | 920 | 4,600 | 1,825.0 |
| 12 | 7.5 | 920 | 4,600 | 1,825.0 |
| 13 | 7.5 | 920 | 4,600 | 1,825.0 |
| 14 | 7.5 | 920 | 4,600 | 1,825.0 |
| 15 | 6.5 | 610 | 3,050 | 275.0 |

IgG LEVELS IN HEIFER NO. 325

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | $\Delta$ IgG |
|---|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 | 0.0 |
| +1 | 6.0 | 500 | 2,500 | 0.0 |
| 0 | 6.0 | 500 | 2,500 | 0.0 |
| 1 | 6.5 | 610 | 3,050 | 550.0 |
| 2 | 7.5 | 920 | 4,600 | 2,100.0 |
| 3 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 4 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 5 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 6 | 8.5 | 1400 | 7,000 | 4,500.0 |
| 7 | 8.5 | 1400 | 7,000 | 4,500.0 |
| 8 | 8.0 | 1100 | 5,500 | 3,000.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 11 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 12 | 8.0 | 1100 | 5,500 | 2,725.0 |
| 13 | 7.5 | 920 | 4,600 | 1,825.0 |
| 14 | 7.0 | 750 | 3,750 | 975.0 |
| 15 | 7.0 | 750 | 3,750 | 975.0 |

IgG LEVELS IN HEIFER NO. L39

(Control Animal)

| Week | Ringo Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 |
| -1 | 6.0 | 500 | 2,500 |
| 0 | 6.0 | 500 | 2,500 |
| 1 | 6.0 | 500 | 2,500 |
| 2 | 6.0 | 500 | 2,500 |
| 3 | 6.0 | 500 | 2,500 |
| 4 | 6.0 | 500 | 2,500 |
| 5 | 6.0 | 500 | 2,500 |
| 6 | 6.0 | 500 | 2,500 |
| 7 | 6.0 | 500 | 2,500 |
| 8 | 6.0 | 500 | 2,500 |
| 9 | 6.0 | 500 | 2,500 |
| 10 | 6.0 | 500 | 2,500 |
| 11 | 6.0 | 500 | 2,500 |
| 12 | 6.0 | 500 | 2,500 |
| 13 | 6.0 | 500 | 2,500 |
| 14 | 6.0 | 500 | 2,500 |
| 15. | 6.0 | 500 | 2,500 |

IgG LEVELS IN HEIFER NO. 227

(Control Animal)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 6.0 | 500 | 2,500 |
| -1 | 6.0 | 500 | 2,500 |
| 0 | 6.0 | 500 | 2,500 |
| 1 | 6.0 | 500 | 2,500 |
| 2 | 6.0 | 500 | 2,500 |
| 3 | 6.0 | 500 | 2,500 |
| 4 | 6.0 | 500 | 2,500 |
| 5 | 6.5 | 610 | 3,050 |
| 6 | 6.0 | 500 | 2,500 |
| 7 | 6.0 | 500 | 2,500 |
| 8 | 6.0 | 500 | 2,500 |
| 9 | 6.5 | 610 | 3,050 |
| 10 | 6.5 | 610 | 3,050 |
| 11 | 6.5 | 610 | 3,050 |
| 12 | 6.5 | 610 | 3,050 |
| 13 | 6.5 | 610 | 3,050 |
| 14 | 6.5 | 610 | 3,050 |
| 15 | 6.5 | 610 | 3,050 |

APPENDIX 8

IgM AND IgG LEVELS IN PREGNANT ZEBU HEIFERS EXPERIMENTALLY INFECTED WITH TRYPANOSOMA VIVAX DURING THE THIRD TRIMESTER AND THOSE OF THE CONTROLS

IgM LEVELS IN HEIFER NO. 1708

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgM |
|---|---|---|---|---|
| -2 | 5.5 | 85 | 425 | -100.0 |
| -1 | 5.0 | 60 | 300 | -225.0 |
| 0 | 5.5 | 85 | 425 | -100.0 |
| 1 | 6.0 | 125 | 625 | 100.0 |
| 2 | 7.0 | 260 | 1,300 | 775.0 |
| 3 | 7.0 | 260 | 1,300 | 775.0 |
| 4 | 8.0 | 520 | 2,600 | 2,075.0 |
| 5 | 8.0 | 520 | 2,600 | 2,075.0 |
| 6 | 8.0 | 520 | 2,600 | 2,075.0 |
| 7 | 8.0 | 520 | 2,600 | 2,075.0 |
| 8 | 8.0 | 520 | 2,600 | 2,075.0 |
| 9 | 8.0 | 520 | 2,600 | 2,075.0 |
| 10 | 8.0 | 520 | 2,600 | 2,075.0 |
| 11 | 8.0 | 520 | 2,600 | 2,175.0 |
| 12 | 8.0 | 520 | 2,600 | 2,175.0 |
| 13 | 8.0 | 520 | 2,600 | 2,175.0 |
| 14 | 7.5 | 370 | 1,850 | 1,425.0 |
| 15 | 7.5 | 370 | 1,850 | 1,325.0 |

IgM LEVELS IN HEIFER NO. 1592

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgM |
|---|---|---|---|---|
| -2 | 7.0 | 260 | 1,300 | 775.0 |
| -1 | 6.5 | 185 | 925 | 400.0 |
| 0 | 6.5 | 185 | 925 | 400.0 |
| 1 | 7.0 | 260 | 1,300 | 775.0 |
| 2 | 7.0 | 260 | 1,300 | 775.0 |
| 3 | 7.0 | 260 | 1,300 | 775.0 |
| 4 | 8.0 | 520 | 2,600 | 2,075.0 |
| 5 | 8.0 | 520 | 2,600 | 2,075.0 |
| 6 | 8.0 | 520 | 2,600 | 2,075.0 |
| 7 | 8.0 | 520 | 2,600 | 2,075.0 |
| 8 | 8.0 | 520 | 2,600 | 2,075.0 |
| 9 | 8.0 | 520 | 2,600 | 2,075.0 |
| 10 | 8.0 | 520 | 2,600 | 2,075.0 |
| 11 | 8.0 | 520 | 2,600 | 2,075.0 |
| 12 | 8.0 | 520 | 2,600 | 2,175.0 |
| 13 | 8.0 | 520 | 2,600 | 2,175.0 |
| 14 | 8.0 | 520 | 2,600 | 2,175.0 |
| 15 | 8.0 | 520 | 2,600 | 2,075.0 |

IgM LEVELS IN HEIFER NO. 1856

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgM |
|---|---|---|---|---|
| -2 | 6.0 | 125 | 625 | 100.0 |
| -1 | 6.0 | 125 | 625 | 100.0 |
| 0 | 6.0 | 125 | 625 | 100.0 |
| 1 | 7.0 | 260 | 1,300 | 775.0 |
| 2 | 8.0 | 520 | 2,600 | 2,075.0 |
| 3 | 8.0 | 520 | 2,600 | 2,075.0 |
| 4 | 8.0 | 520 | 2,600 | 2,075.0 |
| 5 | 8.0 | 520 | 2,600 | 2,075.0 |
| 6 | 8.0 | 520 | 2,600 | 2,075.0 |
| 7 | 8.0 | 520 | 2,600 | 2,075.0 |
| 8 | 8.0 | 520 | 2,600 | 2,075.0 |
| 9 | 8.0 | 520 | 2,600 | 2,075.0 |
| 10 | 8.0 | 520 | 2,600 | 2,075.0 |
| 11 | 8.0 | 520 | 2,600 | 2,075.0 |
| 12 | 7.5 | 370 | 1,850 | 1,425.0 |
| 13 | 7.5 | 370 | 1,850 | 1,425.0 |
| 14 | 7.0 | 260 | 1,300 | 875.0 |
| 15 | 7.0 | 260 | 1,300 | 875.0 |

IgM LEVELS HEIFER NO. 402

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | ▱ IgM |
|---|---|---|---|---|
| -2 | 6.0 | 125 | 625 | 100.0 |
| -1 | 6.0 | 125 | 625 | 100.0 |
| 0 | 6.0 | 125 | 625 | 100.0 |
| 1 | 6.0 | 125 | 625 | 100.0 |
| 2 | 7.0 | 260 | 1,300 | 775.0 |
| 3 | 8.0 | 520 | 2,600 | 2,075.0 |
| 4 | 8.0 | 520 | 2,600 | 2,075.0 |
| 5 | 8.0 | 520 | 2,600 | 2,075.0 |
| 6 | 8.0 | 520 | 2,600 | 2,075.0 |
| 7 | 8.0 | 520 | 2,600 | 2,075.0 |
| 8 | 7.5 | 370 | 1,850 | 1,325.0 |
| 9 | 7.5 | 370 | 1,850 | 1,325.0 |
| 10 | 7.0 | 260 | 1,300 | 775.0 |
| 11 | 7.0 | 260 | 1,300 | 775.0 |
| 12 | 6.0 | 125 | 625 | 200.0 |
| 13 | 5.0 | 60 | 60 | 365.0 |
| 14 | 5.0 | 60 | 60 | 465.0 |

IgM LEVELS IN HEIFER NO. 1671

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration mg/100ml |
|---|---|---|---|
| -2 | 5.5 | 85 | 425 |
| -1 | 5.5 | 85 | 425 |
| 0 | 5.5 | 85 | 425 |
| 1 | 5.5 | 85 | 425 |
| 2 | 5.5 | 85 | 425 |
| 3 | 5.5 | 85 | 425 |
| 4 | 5.5 | 85 | 425 |
| 5 | 5.5 | 85 | 425 |
| 6 | 5.5 | 85 | 425 |
| 7 | 5.5 | 85 | 425 |
| 8 | 5.5 | 85 | 425 |
| 9 | 5.5 | 85 | 425 |
| 10 | 5.5 | 85 | 425 |
| 11 | 6.0 | 125 | 625 |
| 12 | 5.5 | 85 | 425 |
| 13 | 5.5 | 85 | 425 |
| 14 | 5.5 | 85 | 425 |
| 15 | 5.5 | 85 | 425 |

IgM LEVELS IN HEIFER NO.1674

(Control Heifer)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 6.0 | 125 | 625 |
| -1 | 6.0 | 125 | 625 |
| 0 | 6.0 | 125 | 625 |
| 1 | 6.0 | 125 | 625 |
| 2 | 6.0 | 125 | 625 |
| 3 | 6.0 | 125 | 625 |
| 4 | 6.0 | 125 | 625 |
| 5 | 6.0 | 125 | 625 |
| 6 | 6.0 | 125 | 625 |
| 7 | 6.0 | 125 | 625 |
| 8 | 6.0 | 125 | 625 |
| 9 | 6.0 | 125 | 625 |
| 10 | 6.0 | 125 | 625 |
| 11 | 5.5 | 85 | 425 |
| 12 | 5.5 | 85 | 425 |
| 13 | 5.5 | 85 | 425 |
| 14 | 5.5 | 85 | 425 |
| 15 | 6.0 | 125 | 625 |

IgG LEVELS IN HEIFER NO. 1708

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgG |
|---|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 | 0.0 |
| -1 | 7.0 | 750 | 3,750 | 0.0 |
| 0 | 7.0 | 750 | 3,750 | 0.0 |
| 1 | 7.0 | 750 | 3,750 | 0.0 |
| 2 | 7.5 | 920 | 4,600 | 850.0 |
| 3 | 7.5 | 920 | 4,600 | 1,200.0 |
| 4 | 8.0 | 1100 | 5,500 | 1,750.0 |
| 5 | 8.0 | 1100 | 5,500 | 1,750.0 |
| 6 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 7 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 8 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 11 | 7.0 | 750 | 3,750 | 300.0 |
| 12 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 13 | 7.5 | 750 | 3,750 | 700.0 |
| 14 | 7.5 | 750 | 3,750 | 700.0 |
| 15 | 7.5 | 750 | 3,750 | 700.0 |

IgG LEVELS IN HEIFER NO. 1592

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgG |
|---|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 | 0.0 |
| -1 | 7.0 | 750 | 3,750 | 0.0 |
| 0 | 7.0 | 750 | 3,750 | 0.0 |
| 1 | 7.5 | 920 | 4,600 | 850.0 |
| 2 | 7.5 | 920 | 4,600 | 1,200.0 |
| 3 | 7.5 | 920 | 4,600 | 1,200.0 |
| 4 | 7.0 | 750 | 3,750 | 0.0 |
| 5 | 7.0 | 750 | 3,750 | 0.0 |
| 6 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 7 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 8 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 11 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 12 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 13 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 14 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 15 | 8.0 | 1100 | 5,500 | 2,450.0 |

IgG LEVELS FOR HEIFER NO. 1856

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | Δ IgG |
|---|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 | 0.0 |
| -1 | 7.0 | 750 | 3,750 | 0.0 |
| 0 | 7.0 | 750 | 3,750 | 0.0 |
| 1 | 7.0 | 750 | 3,750 | 0.0 |
| 2 | 7.5 | 920 | 4,600 | 850.0 |
| 3 | 7.5 | 920 | 4,600 | 1,200.0 |
| 4 | 7.5 | 920 | 4,600 | 850.0 |
| 5 | 7.5 | 920 | 4,600 | 850.0 |
| 6 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 7 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 8 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 9 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 10 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 11 | 8.0 | 1100 | 5,500 | 2,100.0 |
| 12 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 13 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 14 | 8.0 | 1100 | 5,500 | 2,450.0 |
| 15 | 8.0 | 1100 | 5,500 | 2,450.0 |

IgG LEVELS IN HEIFER NO. 402

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Conc. (mg/100ml) | △ IgG |
|---|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 | 0.0 |
| -1 | 7.0 | 750 | 3,750 | 0.0 |
| 0 | 7.0 | 750 | 3,750 | 0.0 |
| 1 | 7.0 | 750 | 3,750 | 0.0 |
| 2 | 7.0 | 750 | 3,750 | 0.0 |
| 3 | 7.0 | 750 | 3,750 | 350.0 |
| 4 | 7.5 | 920 | 4,600 | 850.0 |
| 5 | 7.5 | 920 | 4,600 | 850.0 |
| 6 | 7.5 | 920 | 4,600 | 1,200.0 |
| 7 | 7.5 | 920 | 4,600 | 1,200.0 |
| 8 | 7.5 | 920 | 4,600 | 1,550.0 |
| 9 | 7.5 | 920 | 4,600 | 1,500.0 |
| 10 | 7.0 | 750 | 3,750 | 700.0 |
| 11 | 6.5 | 610 | 3,050 | 350.0 |
| 12 | 6.5 | 610 | 3,050 | 0.0 |
| 13 | 6.0 | 500 | 2,500 | 550.0 |
| 14 | 5.0 | 335 | 1,675 | 1,375.0 |

IgG LEVELS IN HEIFER NO. 1671

(Control Animal)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration (mg/100ml) |
|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 |
| -1 | 7.0 | 750 | 3,750 |
| 0 | 7.0 | 750 | 3,750 |
| 1 | 7.0 | 750 | 3,750 |
| 2 | 7.0 | 750 | 3,750 |
| 3 | 7.0 | 750 | 3,750 |
| 4 | 7.0 | 750 | 3,750 |
| 5 | 7.0 | 750 | 3,750 |
| 6 | 7.0 | 750 | 3,750 |
| 7 | 7.0 | 750 | 3,750 |
| 8 | 6.5 | 610 | 3,050 |
| 9 | 6.5 | 610 | 3,050 |
| 10 | 6.5 | 610 | 3,050 |
| 11 | 6.5 | 610 | 3,050 |
| 12 | 7.0 | 750 | 3,750 |
| 13 | 6.5 | 610 | 3,050 |
| 14 | 6.5 | 610 | 3,050 |
| 15 | 6.5 | 610 | 3,050 |

IgG LEVELS IN HEIFER NO. 1674
(Control Animal)

| Week | Ring Diameter (mm) | Concentration (mg/100ml) | Final Concentration mg/100ml |
|---|---|---|---|
| -2 | 7.0 | 750 | 3,750 |
| -1 | 7.0 | 750 | 3,750 |
| 0 | 7.0 | 750 | 3,750 |
| 1 | 7.0 | 750 | 3,750 |
| 2 | 6.5 | 610 | 3,050 |
| 3 | 6.5 | 610 | 3,050 |
| 4 | 7.0 | 750 | 3,750 |
| 5 | 7.0 | 750 | 3,750 |
| 6 | 6.5 | 610 | 3,050 |
| 7 | 6.5 | 610 | 3,050 |
| 8 | 6.5 | 610 | 3,050 |
| 9 | 6.5 | 610 | 3,050 |
| 10 | 6.5 | 610 | 3,050 |
| 11 | 7.0 | 750 | 3,750 |
| 12 | 7.0 | 750 | 3,750 |
| 13 | 6.5 | 610 | 3,050 |
| 14 | 7.0 | 750 | 3,750 |
| 15 | 7.0 | 750 | 3,750 |