BADEGGI JOURNAL OF AGRICULTURAL RESEARCH AND ENVIRONMENT, 2024, 06(02), 89-106

NCRI Press

Available online: www.ncribjare.org
ISSN: 2695-2122, e-ISSN: 2695-2114
DOI: https://doi.org/10.35849/BJARE202402/185/009
Journal homepage: www.ncribjare.org

Research Article

The Biology, Epidemiology, Host Variability and Management of Sorghum Smut Diseases in Nigeria: A Review

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Abstract
Sorghum Smut diseases in Nigeria are widely circulated and are economically important in most of the regions of the country growing Sorghum. Perhaps, all the types (4) of smut can co-exist on the same field and are co-extensive with sorghum cultivation. The four smut diseases are incited by four species belonging to two genera of the order ustilaginales, namely; *Sporisorium* or *Sphacelotheca* and *Tolyposporium*. The distribution of the four smut diseases depend on several interacting factors of epidemiology and pathogenesis. It has been reported that all the four species that cause smuts in sorghum have narrow host range. As a consequence, therefore, they do not vary remarkably in their biology and pathogenesis. However, their severity, distribution and prevalence depend on the environmental conditions, seasons and variety of sorghum. Several control as well as management strategies have been suggested for the prevention and control of sorghum smuts including the pre-sowing seed treatment using fungicides, the use of resistant plant variety and modification of cultural and handling practices. However, only few of these measures have been found suitable and promising. Moreover, there is scarcity of scientific information on the incidence of sorghum smuts probably because they were thought to have been eradicated or that they are economically less important in most sorghum growing areas in Nigeria. But with the current climatic changes coupled with the negligence from the poor resource sorghum farmers, sorghum smut diseases are re-emerging with high threshold as such they are becoming very important in Nigeria's sorghum growing regions. This paper reviews the epidemiology, biology and variability of smut pathogens and their possible control strategies. In conclusion, Sorghum smut diseases in Nigeria vary by year, location, and variety, and can be controlled through seed treatment with systemic fungicides and cultural practices.

Keywords: *sorghum smuts diseases; epidemiology; host variability, biology; control*

Introduction
The most common deteriorative fungal diseases affecting sorghum productivity are smuts, downy mildew, ergot, anthractnose, grey leaf sport and oval leaf sport (Singh, 1998; Kutama *et al.*, 2012a). Of these diseases, sorghum smuts are the most important and widespread throughout the world. In Nigeria, smuts are damaging to both local and improved cultivars especially where untreated seeds are sown (Gwary *et al.*, 2007; IPM, 2008; Kutama *et al.*, 2012a). Smut diseases are therefore a constraining factor in sorghum production hence, a threat to the food security in West Africa (Louis *et al.*, 2007). In Nigeria and the world over, four different smut diseases have been identified; they are covered kernel smut (*Sporisorium sorghi*, synonym*: Sphacelotheca sorghi)*; loose kernel smut (*Sporisorium cruentum* synonym*: Sphacelotheca cruenta);* head smut (*sporisorium reilianum* synonym*: Sphacelotheca reiliana)* and long smut (*Soroposporium ehrinbergii* synonym *Tolyposporium ehrenbergiii)* (Frowd, 1980; Marley *et al.*, 2002a and b; Gwary *et al.*, 2007).

Received on 7th July, 2024; Revised on 9th August, 2024; Accepted on 18th August, 2024 and Published on 30th August, 2024

In Nigeria, annual crop losses from all the four smuts were earlier estimated at more than 7-20% of the crop (Kutama *et al*., 2012a, and b), which in some cases yield losses can reach 80% in severely infected sorghum fields (Prom *et al*., 2024). This is however dependent on the region and season as well as other environmental factors prevailing in the field. In some regions and seasons, outbreak of the disease can cause up to 50% - 100% yield loss, although this is rear (Frowd, 1980; Sundaram, 1980). The objective of this paper is to review some aspects of the host –pathogen variability, epidemiology and management practices of sorghum smut diseases in Nigeria with a view to update knowledge of the pathogens' distribution, race, and life cycle and to inform the agricultural sector and the resource-poor farmers of the current situation of the diseases.

Biology of Smut Pathogens

Smut fungi are one of the most important groups of plant pathogens, responsible for quiet dramatic yield losses in cereals (Thomas and Menzies, 1997). They comprise of more than 1500 species and their hosts are distributed all over the whole system of angiosperms (Kutama *et al*., 2010a, 2011b). Smut fungi, belonging to the Ustilaginomycetes and Microbotryales within the Basidiomycota, are parasitic to herbaceous plants like Poaceae and Cyperaceae (Teodor *et al*., 2023). Initially classified in Ustilaginales and Tilletiales, these fungi were traditionally grouped in Heterobasidiomycetes due to their unique life cycle involving basidiospores germinating via hyphae, secondary spores, or budding, and featuring a saprophytic yeast stage (Steins *et al*., 2023). Recent DNA sequence data from the ITS region confirmed the phylogenetic relationships among graminaceous smut genera like Ustilago and *sporisorium*, causing smut diseases in sorghum (Fu *et al*, 2022). The genomic analysis of *Pseudozyma* and parasitic Ustilaginales species revealed conserved genes for mating and meiosis, challenging the assumption of asexual lifestyles in some lineages and highlighting the maintenance of key sexual functions in these fungi (Steins *et al*., 2023).

Sorghum Covered Smut is essentially caused by *S. sorghi* (syn. *Spacelotheca sorghi*) Clinton. It occurs in virtually all sorghum growing areas of the world. In Nigeria, covered smut used to be the most popular and common in most sorghum growing parts of northern Nigeria (Kutama *et al*., 2009a). The fungus attacks almost all plant species belonging to sorghum or graminae (grasses). It is a major disease of sorghum affecting the kernels, more particular the seeds. The disease is also the most destructive of all the smuts because its severity directly depends on the incidence of the disease. In Nigeria, covered smut disease has been reported from almost all areas producing sorghum extensively either for seed or forage value (Kutama *et al*., 2011b).

Loose smut of sorghum is induced by *S. cruenta*. *S. cruenta* (syn *Spacelotheca cruenta*) Kuhn Potter was firstly described as *Ustilago cruenta* by Kuhn in Germany in 1872. Later in 1912, Potter observed and reviewed the literature and described the fungus as *Sphacelotheca* or *S. cruenta* (Kuhn Potter). Ainsworth (1963) further classified the fungus and placed it under the genus *Sporisorium* (with the synonym *Sphacelotheca*). However, *S. cruenta* (Kuhn) Potter is in many respects similar to *S. sorghi*, causing covered kernel smut; and there appears to be no hard and fast dividing line between the two species (Kutama *et al*., 2012b). Kutama *et al*. (2010a, b) had studied some aspects of the biology of *S. cruenta* and that of *S. reilianum*, the incitants of loose and head smuts, respectively. They demonstrated that the fungus, *S. cruenta* causing loose smut in sorghum produces hard resistant spore at the end of the host (sorghum) growing period, called chlamydospores. Spores of *S. cruenta* therefore are formed in the floral bracts or in the ovaries and are called chlamydospoes or more specifically teliospores as demonstrated by Singh (1998). The spores when formed occur in long round; irregular clumps but later separate (Mehrotra and Aggarwal, 2003). They are usually round, or shortly elliptical (Tarr, 1962, Singh, 1998), dark brown, with echinulate wall and measures between 5-10µ x 7-8µ in diameter, hence are larger than that of *S. sorghi*, causing covered smut (Singh, 1998). The spores are dispersed at maturity usually leaving behind the columella. In some hosts varieties, the wall covering the sorus ruptures early while in others it persists or sometimes showing characters of both loose and covered smuts. Germination of the spore occurs in water and can also take place in nutrients agar hence the pathogen can be cultured on artificial medium (Singh, 1998). On germination, the spore produces a tubular structure called promycelium which is of about 4 cells thick. The promycelium produces sporidia which is usually unicellular, hyaline and of about 10-15 µm long (Tarr, 1962).

On the other hand, head smut is incited by *Sporisorium reiliana* (Kuhn) Langdon and Fullerton (syn. *Sphacelotheca reiliana* (Kuhn) Clinton and *S. reilianum* (Kuhn) McAlpine (Singh, 1998). It was identified about 130 years ago by Kuhn when he collected a specimen of the fungus in 1868 from Egypt. The fungus was first recognized in maize as *Ustilago pulveracea* in 1876. After noting that the clamydospores of the fungus tended to cling together in loose impermanent spore balls, McAlphine in 1910 suggested *Sorosporium reilianum* (Kuhn) McAlphine. However, the commonly used name nowadays is *Sphacelotheca reilianum* (Kuhn) Clinton until recently when the whole genus was re-named as *Sporisorium* and the fungus therefore is now called *Sporisorium reilianum* (Tarr, 1962). The fungus causes head smut in maize and in sorghum (Tarr, 1962; Sundaram, 1980; Frowd, 1980; Singh, 1998; Gwary and Asala, 2006; Gwary *et al*., 2007). Similar to its generic partner, *S. reilianum* produces teliospores. The spores of *S. reilianum* are usually distinct from that of *S. cruenta* and *S. sorghi*. They are usually occurring as spore mass, reddish brown, finely or densely echinulate (Kutama *et al*., 2010; 2012; Lynch *et al*., 1988; Mehrotra and Aggarwal, 2003), irregular or more or less spherical in shape and about 9-12 µm in size. The spores form anger ball of about 60-180 at early stage which eventually break up at maturity before spores germinate to produce promycelium of 4-celled thickness, straight or sometime branched bearing sporidia at the terminal portion near the septa. Sporidia are uni-nucleated, but may also be bi-or multinucleated (Teferi *et al.,* 1989).

Long smut, caused by *Tolyposporium ehrinbergii* appears to be the most unpopular smut of sorghum in most areas of sorghum cultivation of the world because of its low economic importance. Long smut pathogen is the only sorghum smut pathogen that belongs to a different genus (*Tolyposporium*) and therefore the only “odd” in the family of smut fungi (Kutama *et al.,* 2022). In the modern classification, some authors classified this pathogen as *Sporisorium ehrinbergii.* Long smut disease, caused by the fungus *Sporisorium ehrenbergii*, is a significant threat to sorghum productivity and food security (Prom *et al*., 2007). The disease can lead to considerable yield losses, particularly in regions where untreated seed is planted (Mahmoud *et al*., 2018). Certain sorghum lines and hybrids have been found to exhibit low levels of infection, suggesting potential sources of resistance (Prom *et al*., 2007, Prom *et al*., 2014). However, the response to long smut disease can vary depending on the environmental conditions and the specific sorghum cultivar (Mahmoud *et al*., 2018). In Nigeria, various complex factors were known to influence the epidemy of sorghum long smut disease both spatial and temporal. Long ago, Marley (1999) discovered that Long smut tends to be most widespread and prevalent in the Sahel savanna region, where the environmental conditions and agricultural practices create a conducive environment for the disease to thrive. This contradicts Pande (1993) who observed that, the incidence of Long smut in Sorghum fields within the sahelian zone was relatively low, with less than or equal to 10% of the fields exhibiting signs of the disease. The persistence of Sorghum long smut in the sahel savanna agro-ecological zone of Nigeria between 1993 to 1999 is highlighted by the presence of the disease in more than 20% of Sorghum fields, indicating its ongoing significance for farmers (Samuel *et al*., 2020). Various management strategies have been employed by farmers, including the use of indigenous knowledge such as field spraying with goat faeces slurry and the placement of neem and Blumea leaves in corn granaries (Hadiza *et al*., 2021; Samuel *et al*., 2020). Additionally, studies have shown that seed treatments with fungicides like tebuconazole can effectively reduce disease incidence caused by *Sporisorium reilianum*, the pathogen responsible for head smut in sorghum (Zhang *et al*., 2021). These findings underscore the importance of continuous monitoring and integrated disease management practices to mitigate the impact of sorghum diseases like long smut in the region (Pande, 1993).

Sorghum long smut exhibits variation across different regions of Nigeria, thereby highlighting the influence of geographical location on the incidence and severity of this disease (Kutama *et al*., 2010b). Recently for instance, within the Sudan Savanna region, Prom *et al*. (2023) revealed a significantly higher occurrence of long smut on the *Warawarabashi* sorghum variety. While on the other hand, in the Borno State, Gwary *et al*. (2009) observed that the incidence of long smut did not exhibit any significant difference between treated and untreated seeds. This suggests that the effectiveness of treatment methods may vary in different regions. It can be correlated that, in the regions of Dosso and Zinder of Niger, which have similar geography with the Sahel regions of some North-Western states of Nigeria such as Borno, Gwary *et al*. (2007) reported a severity of 29% and 100%, respectively, indicating a substantial difference in the prevalence and intensity of long smut depending on the specific region within Nigeria. Furthermore, this evident finding supports the regional and varietal variations in the prevalence of Long smut in Nigeria, emphasizing the importance of considering these factors when developing strategies for disease management and control. This however, highlight a strong need of devising effective measures that could take into account these variations and aim to mitigate the impact of Long smut on sorghum production in Nigeria.

Moreover, a comprehensive survey conducted by Kutama *et al*. (2010) in the Dawakin-kudu local government area of Kano state, Nigeria, demonstrated that smut displayed the highest incidence among sorghum diseases, as it was found in all the 20 locations surveyed. This pervasive presence of smut in the region signifies its status as the most prevalent sorghum disease within this particular area. Therefore, it can be inferred and further support that the overall prevalence and severity of long smut are subject to significant regional variations within Nigeria. Therefore, these regional differences is pivotal for implementing effective disease management strategies tailored to the specific needs of each region; as comprehensive understanding of the factors contributing to these variations and interventions to mitigate the impact of long smut on sorghum cultivation in Nigeria is still less understood. Although, seed treatment with Apron Star and foliar application of Neem seed oil were found to reduce the incidence and severity of smuts in sorghum, whereas emulsified neem seed oil spray regime did not significantly affect smut control (Bdliya *et al*., 2010).

Symptomatology

Covered smut incited by *S. sorghi* is usually characterized by all of the seeds in an affected head been spoiled and superceded by dark brown, powdered mass of spores called Teliospores or Chlamydospores usually held together by a tough white-gray or brown membrane. During harvest, the microscopic spores (4-7µ) are released as a result of the rupturing of the membrane which adhere to the surface of healthy seeds where they over-winter. Therefore, seed transmission of the pathogen is the most essential means of transmitting the disease. When a smut infected seed is sown, the teliospore (4-7 µ) germinate together with the seed there by forming a 4 - celled promycelium (epibasidium) bearing lateral sporidium. The sporidium germinates and infect the developing sorghum seedling. Once in the seedling the fungus grow systemically without causing any damage until during headings. During heading, the teliospores replace kernels and are surrounded by membrane. At maturity the membrane rupture releasing the teliospores to contaminates the seeds or soils. However, teliospores germination depends on the temperature, soil moisture and soil temperature.

On the contrary, plants infected by *S. cruenta* exhibit symptoms of loose smut, such as being stunted with slender stalks and earlier emergence of heads compared to healthy plants (Tarr, 1962; Singh, 1998; IPM, 2008; Mehrotra and Aggarwal, 2003). Additionally, these infected plants may display an increased production of branches or tillers (ICRISAT, 1978; Singh, 1998). The ears are typically produced earlier than usual and infected heads are characterized by their looser, bushier appearance and darker green color, likely a result of glume hypertrophy induced by the pathogen. While all spikelets of an infected head are usually affected by smut, some may manage to escape or proliferate (Tarr, 1962). The head always appears bushy due to the premature rupture of the soil covering membrane, releasing a dark powder of spores (Singh, 1998). Consequently, in cases of loose smut, plants under attack by *S. cruenta* tend to head prematurely, sometimes up to 2 weeks earlier than normal, as the fungus accelerates the growth cycle of the stem while suppressing the infection (John, 1962). The tillers are typically short, slender, and result in an overall stunted plant. A key distinction between covered and loose smuts lies in the fact that plants affected by loose smut exhibit stunted growth, thin stalks, and earlier head emergence compared to healthy plants. Furthermore, it has been documented that both covered and loose smuts involve a phenomenon known as "partial infection," as evidenced by various researchers (Tarr, 1962; Sundaram, 1980; Kutama *et al*., 2011). It is also known that when partial infection occurs in loose and covered smuts, the non-smutted florets produce grains probably because they are morphologically and pathologically similar. The grains produced by the non-smutted florets in loose smut has been experimentally proven by Kutama *et al*. (2012) to contain no any spore or mycelium, hence are healthy.

However, head smut symptoms caused by *S. reilianum* are found in maize and sorghum with some little, but wide differences. In sorghum affected plants *S. reilianum* produces relatively taller plants than healthy ones thereby suppressing that of the floral panicle, (Lynch *et al*., 1980; Singh, 1998; IPM, 2008). The head is typically completely covered with distinctive clusters of small, rolled leaves known as witches brooms, which emerge from the top of offshoots at points or connections, thus the top portion affected by smut is concealed by a dense white layer. It has also been shown that smutted plant have weak root systems and commonly shows more severe attack and susceptibility to stalk and root infection than other healthy plants (IPM, 2008). The most significant decreases were observed in the sections of the plant stem closest to the reproductive structure, with a more pronounced impact on plants naturally infected compared to those deliberately inoculated. Plants that were less impacted exhibited panicles that were unable to produce grains, while panicles that were eventually infected with smut showed abnormal leaf-like growths that developed into leafy shoots (Matheussen *et al*., 1991). However, in maize the disease possess variety of symptoms (Jackson, 2001; John 1962; Singh, 1998; Lynch, *et al*., 1980, 1995).

Physiological Specialization and Races

Smut fungi share a complex life cycle characterized by distinct morphological stages adapted to their parasitic lifestyle on host plants. The initiation phase involves haploid sporidia fusion forming dikaryotic hyphae that penetrate the host plant (Yan *et al*., 2016). This process is mediated by mating type loci signaling pathways critical for pathogenic development as seen in *U. maydis* and *S. scitamineum* (Banuett, 1992). Notably, dual-color imaging systems have been employed to visualize these early interactions in *S. scitamineum*, demonstrating conjugation tube formation leading to dikaryotic filament development within hours post-mating (Yan *et al*., 2016).

Differences in reaction to smut have been observed on the different varieties of sorghum at different locations of Nigeria (Tyagi, 1980). According to Orji *et al*. (1990), the morphological variations existing in the smut fungi might be one of the factors contributing to such differences of races in smut fungi. There are considerable experimental and field evidences of the existence of races in smut fungi. Two physiological races of *S. cruenta* are known and probably others exist (Tarr, 1962). Earlier report has shown that up to three races existed. Frowd (1980) suggested that the existence of three races in *S. cruenta* was probably due to hybridization with *S. sorghi*, the incitant of covered kernel smut. Natural hybridization between *S. sorghi* and *S. cruenta* was very possibly (Mehrotra and Aggarwal, 2003) because of structural and pathogenic similarities that exist between the two pathogens.

On the other hand, experimental as well as field evidences have demonstrated the existence of two major physiologic races of *S. reilianum*, one known to attack maize and the second on sorghum (Tarr, 1962; Frowd, 1980; Jackson, 2001; IPM, 2008). Tarr (1962) presented findings indicating that a mild infection of sorghum with head smut can be observed when sorghum is inoculated with maize head smut. It was discovered that there is a limited capability for head smut to be transmitted between maize and sorghum in both directions. For instance, Al-Sohaily *et al*. (1963) identified four races within the sorghum group by evaluating 18 head smut collections from the USA and India against five sorghum differentials. The emergence of these novel virulent genes in *S. reilianum* is a matter of apprehension in the United States and other regions (Frowd, 1980). King (1972) suggested that a new race is thought to possibly occur in Nigeria probably due to race subdivision. Physiological race within the species may be determined by the differential disease response of various host cultivars (Appel and Gordon, 1998).

Disease Host Range

At present level of knowledge, fungal pathogens classified as smut fungi (including Ustilaginomycetes and Microbotryales, Urediniomycetes) within the Basidiomycota phylum have been identified as parasites of various plant species, particularly herbs from the Poaceae and Cyperaceae families, resulting in significant economic damage to agricultural crops (Bauer *et al*., 1997, 2001; Piepenbring, 2001). Smut diseases caused by fungi of the genus *Sporisorium* have their major host plants as cereal crops. Loose smut caused by *S. cruenta* is restricted to the genus *sorghum* (Tarr, 1962; Frowd, 1980; Singh, 1998) although it has been found on some sugar cane varieties in which *Sorghum halepense* was one parent (Frowd, 1980). Apart from these, *S. cruenta* posses very narrow host range and is therefore economically important only in sorghum, causing a lot of loss in grower profit. Loose kernel smut, caused by the fungus *Sporisorium cruentum* (synonym *Sphacelotheca cruenta*) is less widespread than covered kernel smut. Loose kernel smut attacks all groups of sorghums, including Johnson grass, although certain varieties in some groups are immune or highly resistant. Sudan grass is usually not infected (IPM, 2008).

Conversely, its generic partner *S. reilianum* causing head smut, appear to occur mainly on maize and sorghum (Sundaram,1980; Lynch *et al*., 2001; Martinex *et al*., 1998; IPM, 2008) including Johnson grass, and Sudan grass. Report has shown that the fungus infects three other genera: *Andropogan*, *Cleistachne* and *Euchlaena*. Halisky (1963) have demonstrated the significance of Sudan grass in margins of a field as an alternative host for the pathogen. Therefore, *S. reilianum* possess wider host range than its counterpart and this may be the reason why it is economically more important than its counterpart.

Sporisorium species exhibit an ability for systemic growth within their hosts; mycelial spread occurs *via* vascular colonization facilitating extensive tissue invasion before reproductive structures (teliospores) are formed (Frantzeskakis *et al*., 2017; Diagne-Leye *et al*., 2013). Teliospore formation marks a transition towards reproduction; these structures undergo meiosis generating new haploid sporidia ready for dispersal and infection initiation on new hosts. An intriguing aspect is the alternative respiratory pathways identified in *Sporisorium reilianum* f.sp *zeae* suggesting adaptations for survival under varied environmental stresses encountered during different lifecycle stages (Mendoza *et al*., 2022). Similar mechanisms could be hypothesized for *S. ehrenbergii* given its close phylogenetic relationship within the smut fungi clade.

Comparative transcriptomics across dimorphic fungi reveal divergent mechanisms underlying morphological transitions essential for pathogenicity; however, common themes emerge such as energy production adjustments during hyphal growth indicating shared evolutionary strategies amongst plant-associated dimorphic fungi including potentially *S. ehrenbergii* (Kijpornyongpan and Aime, 2021). Effector proteins play pivotal roles throughout smut fungus life cycles by manipulating host defenses ensuring successful colonization; examples include SsPEP1 in *S. scitamineum* which affects both fungal development and interaction with sugarcane hosts through inhibition of peroxidase activity - an enzyme involved in reactive oxygen species detoxification within plants (Lu *et al*., 2021a).

Despite advances in understanding specific aspects like genetic regulation of filamentation or effector-mediated host manipulation observed across related species Lu*et al*. (2021b), direct investigations into *Sporisorium ehrenbergii's* lifecycle remain scarce necessitating extrapolations from closely related taxa thereby introducing potential inaccuracies due to interspecies variability.

Distribution of Loose and Head Smuts

Loose kernel smut (*S. cruentum*) is commonly present in all sorghum cultivation regions worldwide (Frederiksen, 1986). The management of this disease can be achieved through the application of specific fungicides like Thiram and Carboxin to treat the seeds. Nevertheless, this practice has not been widely embraced by the target demographic, namely resource-constrained, small-scale farmers in Nigeria. Smuts represent a collective of diseases primarily found in tropical regions where sorghum is a predominant crop used for either sustenance or animal feed. These diseases are recognized as the most significant and prevalent in all sorghum-growing regions of Nigeria, typically causing more harm to indigenous varieties than to enhanced cultivars (Selvaraj, 1980; IPM, 2008; Gwary *et al*., 2007), probably because most of the seed farmers used is that saved and initially infested or occasionally been replenished by contaminated seeds bought from local markets (Singh *et al.*, 1997; Kutama *et al.*, 2010a; Umar *et al*., 2009). In all the sorghum producing areas of Nigeria all the four smuts are found, at times co-extensive with sorghum cultivation (Sundram, 1980). The pattern of distribution of loose and head smuts depends on so many interacting factors of epidemiology and pathogenesis. Omar (1992) demonstrated that the incidence of the smut diseases differs from one region to another and with the season. He demonstrated that loose smut is of particular importance in ratoon and fodder sorghum. The distribution of loose smut in the world probably reflects its seed-borne nature. Although apparently less widespread than head and covered smuts, loose smut has been reported in north America, Jamaica, Europe, Cyprus, Uganda, Kenya, Sudan, Congo, China, India, Palestine, Russia, Manchuria, Italy and Germany (Frowd, 1980). There is however little details on the incidence and spread of loose smut in Nigeria (Frowd, 1980) because most farmers recognized covered smut (King, 1972; Tyagi, 1980; Gwary *et al*., 2007). However, since loose smut is transmitted in the same mechanism and the its pathogen is in many respect identical to that of covered smut often at times being confused with the latter, its distribution may not necessarily differ from covered smut (Omar,1992; Singh,1998). In Nigeria, a study conducted in various sorghum cultivation areas across the four climatic zones of the Nigerian savanna revealed shifts in the frequency, severity, and spread of sorghum smuts. The prevalence of loose smut *S. cruenta* and long smut Tolyposporium ehrinbergii was higher in the Sahel region compared to the Sudan savanna, with rates of 21.8% and 15.5% respectively (Marley and Aba, 1999).

On the same flat form, head smut (*Sporisorium reilianum*) appears in all sorghum and maize growing area around the globe. It has been reported from Asia, Australia, Europe, central and south America, India (Singh,1998; Frowd, 1980), in Canada (Lynch *et al*.,1980), in Nigeria (Selvaraj,1980; Marley and Aba, 1999; Marley *et al*.,2002; Bdliya, 2005, Kutama *et. al.*, 2011a,b,c.; Kutama *et al*., 2013a,b,c,d), in France (Martinex *et al*.,1998), in Texas (Frederiksen and Reyes,1980), Kenya (Ngugi *et al*.,2001; Jackson,2001) and many places elsewhere. A pronounced increase in. the prevalence of sorghum head smut has occurred since the advent of the high-yielding but smut-susceptible grain sorghum hybrids. During 1957-60 head smut was recorded in no fewer than 18 hybrid varieties in California (Halisky and Dale, 1961)**.** In Nigeria, *Sporisorium reilianum* affects sorghum varieties with delayed maturity, which are extensively grown by most of the resource poor farmers. Although incidence may be low, in present years the disease has increased considerably in the Sudan and northern guinea savanna (Marley and Aba, 1999, Kutama *et al*., 2016). Earlier reports revealed that the disease in sorghum used to be greater in parts of the fields near trees, a condition they called ‘’tree effect” (Singh, 1998).

Epidemiology of Smut Diseases

Smut diseases of sorghum are a limiting factor to sorghum production in Nigeria (Kutama *et al*., 2012; Marley and Aba, 1999) and in all sorghum producing areas of the world (Frowd, 1980; Gwary and Asala, 2006; Gwary *et al*., 2007; IPM, 2008). The Four smuts are co-extensive with sorghum production in Nigeria (Frowd, 1980) so that few or no sorghum variety is resistant to all the four smuts. The epidemiology of the different smut diseases differs quite remarkably due to the quarsi-systemic nature of the diseases (Singh, 1998). Several workers opined that individual smut disease is monocyclic because the pathogenesis of most smut diseases involves a single cycle after infection and there is no repetition of the cycle during that season (Singh, 2005). This is contained in an earlier report that in monocyclic disease, only the initial inoculum is effective for the epidemic as no infection from plant to plant occurs (Kutama *et al.*, 2012a,c, 2013a,b,c; Vander Plank, 1963).

Loose smut pathogen, *Sporisorium cruenta* is externally seed-borne (Tarr, 1962; Singh, 1998; Mehrotra and Aggarwal, 2003) but may also be soil-borne in dry regions (Singh, 1998). Tarr (1962) demonstrated that under suitable conditions, however, sorghum heads may become secondarily infected by secondary spores transmitted by air later in the season; in which case the growth of the plant is never affected. Loose smut is of particular importance in ratoon fodder sorghum (Omer, 1995) in which case the disease is therefore a compound interest or polycyclic in nature. In polycyclic pathogens, the cycle is repeated many times in the same season on the host through spore-infection-spore chain (Singh, 2005). Most workers have shown that loose smut, *S. cruenta*, is purely seed-borne and that teliospores in the soil are completely unimportant for its spread (Sundaram, 1980; Singh, 1998, 2005; IPM, 2008; Kutama *et al.*,2012a,b.c). Teliospores germinate along with the seed by first forming a thick, usually 4-celled promycelium bearing lateral sporidia. The sporidia germinates and infect the developing sorghum seedling (Tarr, 1962, Singh, 1998; Carole *et al*., 2002; IPM, 2008; Kutama *et al.*,2013a,b). Seedling infection occurs on a wide variety of soil moisture and soil temperature but it was found that temperature of 68-77^{o}F or 22-25^{o} C is the best (IPM, 2008). However, sequel to seedling infection, there is no any visible symptoms that appear on the seedling. The fungus remains systemically in the plant unobserved until heading, when long pointed smut galls develops in place of normal kernels or grains. The teliospore of *S. cruenta* can stay for years if ungerminated, attached to seeds or in the soil. These spores do not however survive in the soil for many years probably because they germinate when the soil temperature and moisture are optimum (Tarr, 1962; Sundaram, 1980; Singh, 1998). Therefore, the spores over winter only in seeds and is unlikely in the soils. It has also been earlier noted that

On the other hand, head smut incited by *S. reilianum* is potentially a serious disease in most of the sorghum and maize areas of the world. Several researches conducted on the epidemiology of the head smut pathogen have confirmed that the disease is essentially a soil-borne disease in which infection occurs through or at seedling stage. Although seed transmission of the disease appears to occur in maize, there is no any report of its occurrence in sorghum (Tarr, 1962; Singh, 1998). In maize, the disease is characterized with early appearance of chlorotic flecks on the leaf and anthocyanin accumulation on stem (Matyac and Kommedhal, 1985). At maturity however, the ears and the tassels becomes smutted. In contrast, sorghum shows no any yellowishness, perhaps, symptoms are less varied in sorghum (Tarr, 1962). However, in both sorghum and maize, the infected plant grows taller than the healthy ones (Carole *et al*., 2002) probably because the fungus tends to stimulate the growth of the leafy stem and suppresses that of the floral panicles (Tarr, 1962).

Effect of Plant Age

Plant age appear to be important for the initiation of infection by smut pathogens. Most studies indicate that sorghum is susceptible only for a short period during the seedling stage (Metha, 1965). Infection occurs from hyphae arising directly from germinating teliospore (RPD, 1990). Seedling infection occurs when sporidia of opposite mating type fuse with the germinating seedling of the host plant (IPM, 2008). Several interacting factors determine the spread and severity of sorghum smut diseases *vis a vis* sorghum growing regions of the world.

Effect of Host Plant Nutrition

The effect of nutrition on the incidence of smuts has been investigated by many workers. It is generally agreed that excess nitrogen application usually increases the incidence of disease and the disease incidence usually reduces when soil is deficient in phosphorus and nitrogen (Singh, 2005). Conversely, increase in potassium concentration in the soil reduces the incidence of the disease so that potassium confers resistance to diseases in most plants. The effect of varying levels of nitrogen applied as urea in a single dose before planting on grain smuts incidence in a susceptible inoculated local *fara-fara* selection was studied at Kano in 1977. Grain smut was severe in low nitrogen levels (Selvaraj, 1980) and this could be the same with loose smuts since the two pathogens are similar morphologically and pathologically. In agricultural settings, plants cultivated in fields with low quality soils and inadequate management may suffer from significant infestations of covered and loose smuts.

On the other hand, fertility levels have also been reported to increase, decrease or have no effect on the frequency and development of head smut. Head smut incidence decreased when ammonium nitrate, super phosphate, potassium chloride or calcium chloride was applied (Popov, 1968). Smut incidence increased with the application of ammonium sulphate, ammonium nitrate, super phosphate and potassium chloride (Baier, 1962; Frag, 1975). Matyac and Kommedahl (1985) reported significant reduction in the frequency of head smut in corn following applications of urea, ammonium sulphate and triple super phosphate whereas the addition of calcium nitrate caused slight but non-significant disease increase. In a recent *in-vitro* study of some chemicals on the germination and sporulation of *S. reilianum*, magnesium sulphate and potassium chloride were found to significantly increase the germination and growth of the pathogen while zinc nitrate and chlorothalinol significantly inhibited growth. In 2010, Kutama *et al*. (2010a, c) studied the effects of nitrogen fertilization on the incidence of sorghum head smut caused by *S. reilianum*. Results of the study revealed that

Germination and Germination Requirement of Spores

Chlamydospores produced by the smut pathogens readily germinate in water or agar medium *in vitro* (Tarr, 1962; Singh, 1998; Frowd, 1980; Orji *et al*., 1991). In *S. cruenta*, upon sowing the infected seed, teliospores germinate along with the seed through a thick four celled promycelium bearing lateral or terminal unicellular, hyaline sporidia measuring 10-15µm. The sporidia germinate and infect the seedling (Ahlawat, 2007; IPM, 2008). In water, or at high temperature, or both the branched hyphae tend to develop rather than sporidia. In nutrient solution, copious sporulation tends to occur (Tarr, 1962). The four celled promycelium developed from the diploid chlamydospore each believed to have undergone mitosis. Environmental conditions governing infection by *S. cruenta* has been studied. It was established that conditions governing germination in *S. cruenta* are virtually similar to that of *S. sorghi*, causing covered smut and the infection occurs at a temperature 20-25^{o}C with a wide range of humidity (Ahlawat, 2007) and slightly acidic soil (Tarr, 1962; Singh, 1998). The fungus exhibits systemic growth within the plant without displaying any apparent symptoms. Symptomatic manifestations only become apparent upon the emergence of the head. Instead of normal seeds, elongated, black, and pointed smut galls or sori are observed. Thus, the presence of teliospores in the soil is deemed insignificant for the onset of infection (Ahlawat, 2007; IPM, 2008; Singh, 1998).

Upon sowing sorghum seeds, teliospores (9-14µ in diameter) present in the soil undergo germination concurrently with the seeds, resulting in the formation of a four-celled branched promycelium that carries sporidia primarily at the ends near the septa. These sporidia can either sprout by generating yeast-like secondary sporidia or directly germinate through the production of a germtube that penetrates the meristematic tissue of seedlings. The sporidia exhibit quicker germination rates under moist soil conditions at temperatures ranging from 27-31o C. In addition, the infection of seedlings may occur through teliospores that have already adhered to seeds during the previous season. Consequently, healthy soil can become infected through seed contamination. Notably, the role of seed contamination in causing infection appears to be insignificant.

Methods of Artificial Inoculation for the Screening of Sorghum Lines Against Smut Diseases

Many techniques of artificial inoculation in both loose and head smuts have been suggested by many workers (Guo *et al*., 2024; Márquez-Licona *et al*., 2018). It is however apparent that the appropriate inoculation procedure depends on the route of infection of a particular pathogen and the developmental stage of the host plant when it is most susceptible. These plus the question of whether small or large number of sorghum lines are used for the inoculation; partly decide which method mostly fit. Some of the techniques described for artificial inoculation in loose and head smuts are;

Seed Application

In this technique, sorghum seeds are combined with dry teliospore at a dosage of 0.5 g/kg and planted in cold soil as described by Sundaram (1980). This approach has been observed to lead to significant infection in fields planted in cold and moist soil conditions. It is commonly employed for the assessment of sorghum genetic resources and breeding components. This technique however, is used for seed-borne smut pathogens responsible for covered and loose smuts only (Frowd, 1980). The method is not suitable for head and long smuts as neither of them is seed-borne in nature. However, in most screening procedure, the inoculum to seed proportion varies widely between individual tests (Ranganathaian and Govindu, 1970).

Application of Germinating Teliospore to Seeds

Seeds are mixed with germinating teliospores at 0.2% by weight and later sown immediately in cool soil (Selvaraj, 1980). This method may be used for both loose and head smuts but better for head smut.

Placing Germinating Teliospores in the Planting Hole

This technique is employed just before planting. Approximately 0.1 g of germinating teliospores is placed in the planting hole (5-7 cm depth). The use of this method in head smut has produced very good infection results (Kutama *et al*., 2012; 2022).

Hypodermic Injection Technique

Teliospores weighing 0.5 grams, harvested from the previous year, undergo germination in 1 cubic decimeter of distilled water, followed by blending for a duration ranging from half to one minute. Subsequently, a volume of 0.5-1.0 milliliters of the germinating material is injected hypodermically at the growing point of the plant. This process is replicated twice, specifically on the 21st and 40th days post planting, as documented by Frowd (1980) and Sundaram (1980). This methodology has proven effective for combating both loose and head smuts. Keay *et al*. (1969) demonstrated that 64% of sorghum plants aged between 3-4 weeks became infected with loose smut upon inoculation using a hypodermic syringe. In contrast, Kutama *et al*. (2012; 2022) utilized this approach to assess sorghum genotypes' resistance to head and loose smuts.

Cotton Wool Inoculation Technique

Kutama *et al*. (2012) devised a technique wherein seeds of two sorghum varieties were individually soaked in dampened sterilized cotton wool for 48 hours in Petri dishes at room temperature. Subsequently, the germinating seedlings were moved to fresh sterilized dry Petri dishes along with the cotton wool to eliminate excess moisture. These germinating seedlings were then combined with dry teliospores of either *S. cruenta* or *S. reilianum* at a ratio of 10:1 (g seedling: teliospores). The Petri dishes were placed in incubation at 28 ± 2^{o}C for a duration of 5 days. On the sixth day, the seedlings, along with the attached cotton, were transplanted to the field in the evening hours (1600 -1900), being carefully covered with a minimal amount of soil to mature under rainfed conditions. A control group was also established following the same procedure, but without the incorporation of teliospores.

PDA-Seed Inoculation Technique

Seeds of various sorghum varieties were soaked in water for a period of 48 hours. Following this, the germinating seedlings of each variety were placed individually onto a PDA plate. A quantity of 0.1 g teliospore powder from both *S. reilianum* and *S. cruenta* was then introduced to each set of germinating seedlings to induce infection, after which they were incubated at a temperature of 28±2oC for a duration of seven days. Subsequently, the seedlings were carefully transplanted into the field, along with the remaining PDA gel. A control group was also set up, however, without the addition of teliospores

(Kutama *et al*., 2012; 2022).

Assessment of Yield Loss Due to Loose and Head Smuts

The losses resulting from sorghum loose smut in Nigeria are generally minimal, seldom surpassing 10% even in certain highly affected regions (Sundaram, 1980). These losses are contingent upon the specific season and variety of sorghum being grown (Singh, 1998). Nonetheless, in cases of loose smut, the plants that are infested tend to produce very few grains, leading to a reduction in yield that is directly correlated to the extent of plant infestation. King (1972) had earlier reported only 2% yield loss hence considered the disease of fewer problems at that time. Perhaps, farmers do not know much about loose smut but recognize covered smut because of their close symptomatic resemblance which makes the two diseases confusing (Gwary *et al*., 2007). In a more recent report by Marley and Aba (1999), loses due to loose smut was between 5.0 to 25% in Nigeria (Kutama *et al*., 2014). Frowd (1980) provided evidence that in regions where the practice of ratooning sorghum is implemented, loose smut may reach epidemic proportion and causes incredible loss in the economic value of the crop. In Kenya, Ngugi *et al*. (2002) reported *S. cruenta* in 42 to 47 % and 7 to 24 % of the fields surveyed in 1995 and 1996 respectively even though with low incidence. It is apparent that although the incidence per field may be low, the disease prevalence is high and may likely result into higher economic lost.

The incidence of head smut is relatively high in all sorghum and maize growing regions in Africa, especially in low-lying fields (Sundaram, 1980). In Nigeria, the disease is particularly severe along the northern Sudan savanna belt (Sundaram, 1980; Marley and Aba, 1999). Yield losses ranging from 30% to 100% can occur in hotspot areas. In inoculation studies conducted in Victoria and College Station, Texas, when grain sorghum plants were inoculated at varying frequencies to establish different levels of head smut within a constant plant density of 150,000 plants per hectare, a linear relationship between head smut incidence and grain yield was observed. It was estimated that a 1% increase in head smut incidence led to a decrease in yield by 44.5±2.7 kg/ha, which is more significant than the yield losses caused by downy mildew (Frederiksen and Reyes, 1980; Fagwalawa *et al*., 2013). In Kenya, Ngugi *et al*. (2002) reported *S. reilianum* as the most common of the panicle diseases being observed in 73 to 75 % of the field in1995 and 1996 surveys. Frederiksen (1998) obtained disease incidence ranging from 50 to 80 % when sorghum SC241-12E line was grown under field conditions. The high infection level indicates that the disease can cause considerable yield loss in the crop. According to Silveraj (1980) infection incidence of head smut is often greater in parts of the fields near trees. Halisky (1962) stated that significant amount of disease can only results from inoculum in the soil and not from seed-borne infection, which shows that there are variations in the consequent yield losses. The fact that head smut pathogen possess wide physiologic races may explain the reason why there are differences in the yield loss due to the disease. Whatever, it is evident that head smut causes more economic yield losses in both sorghum and maize (Jackson, 1980; Martinez, *et al*. 2000, 2002) than loose smut (Silveraj, 1980; Tyagi, 1980).

Management and Control of Sorghum Smut Diseases

Several management strategies have been suggested by workers for the prevention and control of sorghum smuts. These include; seed treatment using fungicides, use of resistant plant varieties and modification of cultural and handling practices. However, only few of these measures have been found suitable and promising.

Use of Fungicide as Seed Treatment

The use of fungicide in the control of sorghum smuts has been suggested by many researchers (Kutama *et al*., 2012, 2013, 2016, and 2021). The primary objective of seed treatment is to enhance seedling establishment and manage smut. However, the effectiveness of fungicidal treatments in controlling smut diseases has shown considerable variation in both results and efficacy. On a general basis fungicide is used in plant disease control essentially aimed to create a toxic barrier between the host plant and the pathogen and to eradicate the pathogen present at a particular site on or in the host (Singh, 2005; Kutama *et al*., 2012, 2013). Therefore, fungicidal seed treatment could only be effective in the control of seed-borne pathogens (Jerger *et al*., 1998; Zarafi *et al*., 2012; Kutama *et al.*, 2012, 2013). Seed treatment has been found to be very effective in the control of loose and covered smuts but not effective in the control of head and long smuts probably because in the latter, infection of the host via seed does not occur as a result chemical seed treatment will not control the disease (Jacson,1980; Kollo, 2000; Manzo,1976). Although fungicide is effective in the control of loose and covered smuts and many fungal diseases (Okorie *et al*., 2004) its application is not economical (Selvaraj, 1980) and possess problem to the ecosystem and therefore not eco-friendly. Seed treatment therefore that is inexpensive, safe, and easy to apply as well as eco-friendly is the only recommended chemical method for farmers in Nigeria (Selvaraj, 1980).

Kutama *et al*. (2011) reports have shown that seed treatment with formulations containing thiram (Thiram/Aldrin) usually sold as Aldrex T , Thiram and Lindane usually sold as Fernasan D as well as Carboxin were all time practice which give almost 100% control of covered, loose and head smuts in routine seed-dressing trials. Numerous experiments have been undertaken to evaluate the efficacy of fungicides in controlling smut in farmers' fields. The majority of treated fields exhibited infection rates of less than 1%, whereas untreated seeds showed higher levels of infection. Various seed treatments, including Thiram, Maneb, Captan, Mancozeb (Dithane M-45), Quintozene (Brassicol), Aldrex-T, and Fernasan D, were investigated. Seeds subjected to these chemical treatments produced significantly higher stand counts compared to control seeds, and the treatments provided satisfactory control of grain smuts. However, with the advent of systemic fungicides such as metalaxyl (Methyl-N-(2, 6-dimethylphenyl)-N-(methoxyacetyl)-DL-alaminate), a broad approach to seed dressing became realistic and many more systemic fungicides are now discovered. Metalaxyl is a systemic fungicide absorbed through the leaves, stem, and roots, capable of inhibiting protein synthesis in the fungus. It can be administered in various formulations to combat downy mildew in sorghum and pearl millet (Zarafi *et al.*, 2009), smuts and molds (Gwary *et al*., 2007). It is usually traded as Ridomil in liquid form or as Apron plus, Apron star, Apron extra in Nigeria. This fungicide has been found to completely wipe out all smut pathogens that are naturally seed-borne in nature. Other systemic fungicides used in the control of smuts include benomyl (Benlate), Carbendazin (Bavastin), Thiabendazole (Tecto, Carboxin (Vitavax) and Thiophanate methyl (Topsin). Systemic chemicals have been found to control both loose and head smuts as reported by (Kutama *et al*., 2011) in a new approach to loose and head smuts management. However, most of the success of these trials was based on the artificial inoculation technique of the pathogen on to the host (Kutama *et al*., 2011a,c, 2012a,b). It has been reported that there was a general reduction of sorghum head smut following inoculation technique of stem injecting the teliospores and sporidia in the planting hole.

Use of Resistant Variety

Host plant resistance offers a practical, cost-effective, environmentally sustainable, and economical approach to disease control. The use of resistant varieties has likely been the primary method for managing diseases in sorghum (Silveraj, 1980). It is the best control strategy for sorghum smut diseases (Tyagi, 1980; Kumar and Nath, 1991b; Manzo, 1976; Kollo, 2000). This is probably because a few or no sorghum variety or genotype is immune or resistant to the smut diseases (Kollo, 2000, Omer *et al*., 1990). Pastor-Corale and Frederiksen (1980) reported that the use of resistant sorghum germplasm continue to be the single most important method of diseases especially in developing countries where it is cultivated under subsistence levels.

The identification of resistance sources is a slow process due to the challenges in observing disease expression in late-maturing, day-length-sensitive sorghum (Craig and Frederiksen, 1992), as well as various plant and environmental factors that interfere with natural infection events. Osorio and Frederiksen (1998) noted that head smut resistance in sorghum results from the action of one or more distinct host mechanisms. Craig and Frederiksen (1992) described a factor or group of factors acting at the meristematic level, possibly controlled by race-specific resistant genes, which can be identified through inoculation with appropriate pathogen races. Kutama *et al*. (2010a) identified *S. reilianum* as a variable pathogen. Metha (1965) suggested that variations in virulence to sorghum enable the pathogen to overcome genetic resistance in the meristematic type of the host.

Modification of Cultural Practices

Rouging

Of the many cultural practices reported to be effective against smut diseases rouging of infected panicle at or prior to grain filling stage is one of the most feasible, cheap and reliable as well as effective method of controlling smut diseases. Removal of infected plants during the growing season of the crop is a common practice by farmers in northern Nigeria (Emechebe personal communication). Once an infected crop is removed, the possibility of inoculum spread from crop to crop is highly limited as such the chance of secondary infection is significantly reduced (Kutama *et al*., 2022). Earlier report by Tarr (1962) suggested the destruction of Johnson grass including the rhizome and collection and destruction of smutted heads in areas where sorghum is grown for the control of loose and head smuts respectively. The effect of removing sorghum head smuts on the control of the disease was investigated by Bdliya (2005) where a significant reduction in the incidence of the diseases on the on-farm trial and the experimental plots was obtained. Although not much experimental evidences are available on the effect of removal of smutted heads in the control of loose smut, it is apparently practical that once the heads are removed early before grain filling subsequent seed-transmission could be significantly reduced as no teliospores are available to contaminate the healthy seeds. This method may therefore be employed to reduce the incidence of the four smut diseases.

Date of Sowing

In Nigeria, sorghum is usually sown immediately the rain become established probably because most of the landraces cultivated year in year out in most sorghum growing regions are late maturing , requiring about 4-5 months. Early planting typically results in early establishment and, in many cases, higher yields. However, this practice has been associated with severe long and head smut infections (Kutama *et al*., 2012a; 2022). Conversely, when improved early varieties are planted, they often mature before the end of the rainy season, leading to the molding of the heads (Selvaraj, 1980). Therefore, while early sowing of sorghum increased grain yield compared with late planting in some diseases like sorghum downy mildew because of increase in pressure of the inoculum due to conidia production (Anaso *et al*., 1989; Cohen and Sherma, 1977; Kutama *et al*., 2010), in the smut diseases, early sowing increases the incidence of head (Frowd, 1980) and long smuts (Kollo *et al*., 2007). Late sowing in areas with longer period of rainfall may therefore likely be a way of reducing head and long smuts since it can result in the reduction of spore population in the soil.

Conclusion

Sorghum smut diseases occurs in sorghum every year in the Sudan savanna of Nigeria but the incidences of the diseases varies with year/season, locations and sorghum variety. It also appeared that the infective propagules of all the four smut pathogens popularly called teliospore, bear little or no variations in the color of the aggregate and individual spore isolate of virtually all the four smut pathogens. This indicates that the pathogens may likely behave similarly. However, the teliospores possess physiologic race differences with regard to different or the same sorghum varieties. Studies on the epidemiology of the four smuts suggested that they are essentially externally seed-borne in nature and that internal seed transmission of the pathogens is unlikely. As such, all of the four sorghum smut could properly be controlled through seed treatment using systemic fungicides, and culturally by roughing and late planting.

Acknowledgement

The authors are indebted to important persons such as Professor A. M. Emechebe and Professor B. S. Aliyu for their immense supports.

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