Fumonisin B_1 Production by Strains from Different Mating Populations of *Gibberella fujikuroi* (*Fusarium* Section *Liseola*)

John F. Leslie, Ronald D. Plattner, Anne E. Desjardins, and Carla J. R. Klittich

First and fourth authors, associate professor and former postdoctoral research associate, Department of Plant Pathology, Throckmorton Hall, Kansas State University, Manhattan 66506-5502. Second and third authors, National Center for Agricultural Utilization Research, U.S. Department of Agriculture, Agricultural Research Service, 1815 University Street, Peoria, IL 61604. Present address of fourth author: Rhône Poulenc Agro AB, P.O. Box 11555, S-100 61 Stockholm, Sweden.

Supported in part by the Kansas Agricultural Experiment Station and by grants from the Kansas State Board of Agriculture (Kansas Corn Commission and Kansas Grain Sorghum Commission), and by the International Sorghum/Millet Collaborative Research Support Program (INTSORMIL) AID/DAN-1254-G-00-0021-00 from the U.S. Agency for International Development, Washington, DC (Leslie and Klittich).

Contribution 92-35-J from the Kansas Agricultural Experiment Station, Kansas State University, Manhattan.

Accepted for publication 23 October 1991 (submitted for electronic processing).

ABSTRACT

Leslie, J. F., Plattner, R. D., Desjardins, A. E., and Klittich, C. J. R. 1992. Fumonisin B_1 production by strains from different mating populations of *Gibberella fujikuroi* (*Fusarium* section *Liseola*). Phytopathology 82:341-345.

Fungal isolates of *Gibberella fujikuroi* are economically important as plant pathogens and as producers of mycotoxins. The species can be subdivided into six distinct mating populations that probably represent different biological species. Members of four of these mating populations—A, D, E, and F—are commonly found in asymptomatic and diseased maize and sorghum plants. We tested 56 *G. fujikuroi* isolates that had been collected in Kansas and assigned to mating populations for their ability to produce the mycotoxin fumonisin B_1. When grown on maize grain under laboratory conditions, members of the A population could produce an average of 1,786 ppm of the toxin, members of the D population averaged 636 ppm, the E population 33 ppm, and the F population 7.5 ppm. Strain-related variability in fumonisin B_1 production was relatively large in the A and D populations (307–4,425 and 4–2,618 ppm, respectively) and relatively low in the E and F populations (6–146 and 2–35 ppm, respectively). The level of fumonisin B_1 produced by the different isolates did not appear to be related to geographic origin, host, or disease status of the host plant. Our data are generally consistent with previous work on this mycotoxin, but the differences between the A and F mating populations are significant because both of these groups share the *Fusarium moniliforme* anamorph.

Additional keywords: corn, *Fusarium proliferatum, Fusarium subglutinans, Sorghum bicolor, Zea mays.*

Fungal isolates belonging to *Fusarium* section *Liseola* are distributed worldwide on many economically important plants including rice (50), maize (29), sorghum (29), mango (54), pine (9), asparagus (25), pineapple (44), and sugarcane (35). Additionally, some strains produce significant quantities of secondary metabolites, for example, gibberellic acids (40) and mycotoxins such as moniliformin (33,34), fusarin C (55), fusaric acid (34), and fumonisins (12).

The nomenclature used to delimit species within this section is not settled. Macroconidial morphology, the most commonly used trait in distinguishing species of *Fusarium,* is not useful for distinguishing the species within the *Liseola* section (38); consequently, some authorities (e.g., 48,49) have recognized only a single species (usually *F. moniliforme* J. Sheld.) within this section. Nelson et al (38) distinguished four species within *Liseola* based on the presence of monophialides and polyphialides and the presence of microconidia in long chains, short chains, or false heads. Using similar morphological characters, Nirenberg (39) recognized six species within this section. Leslie (26) has taken another approach, using the formation of the sexual stage to distinguish species. In *Gibberella fujikuroi* (Sawada) Ito in Ito & K. Kimura (the perfect stage associated with isolates in *Fusarium* section *Liseola*), six different mating populations (designated by the letters A–F) have been recognized (16,17, 22,24,26). Isolation of the mating populations is prezygotic, and these different populations appear to represent different biological species. Following the taxonomic systems proposed by Nelson et al (38), members of both of the A and F populations are *F. moniliforme,* members of both of the B and E populations are *F. subglutinans* (Wollenw. & Reinking) Nelson, Toussoun & Marasas, and members of the D population are *F. proliferatum* (T. Matsushima) Nirenberg (22,26). These distinctions are important because different mating populations are found preferentially on different hosts (28).

Equine leukoencephalomalacia and hepatosis have a long record of association with maize contaminated with *Fusarium* spp. from the *Liseola* section (1–6,14,18,19,23,31,41,47,57). More recently, leukoencephalomalacia has been found in deer that were believed to have consumed maize contaminated with *F. moniliforme* (15), and this fungus has been correlated with a pulmonary edema syndrome in swine (13,23,45). Fumonisin B_1 induced equine leukoencephalomalacia when administered intravenously (30) or orally (19), and fumonisin B_1 has induced pulmonary edema in swine (13). The presence of this compound also has been correlated with cancer-promoting activity in rats (12). Determinations of toxic levels of the compound and its distribution in commercial feeds have just begun (46,52,56).

Members of *Fusarium* section *Liseola* are widely distributed in commercial feeds and fields, and many of the strains in this section, but not in other *Fusarium* sections (51), have the potential to produce significant levels of fumonisin B_1. Strains of *F. moniliforme* and *F. proliferatum* recovered from feeds associated with equine leukoencephalomalacia produced significant quantities of fumonisin B_1 (45). Geographically diverse strains of *F. moniliforme* collected from various substrates in Africa, Asia, and North America produced significant levels of fumonisin B_1, but other strains, primarily from Africa and Australia, produced little, if any, fumonisin B_1 (37). The nearly universal distribution of these fungi in maize and sorghum (29,32,53) and their ability to be internally seedborne in symptomless, apparently healthy grain (11,32,53) combine to suggest a significant potential for widespread contamination of human foods and animal feeds. In

TABLE 1. Mating type, origin, and fumonisin B_1 production of *Fusarium* strains used in this study

Strain	MT[a]	Female fertile?	Fumonisin B_1 (ppm)	Original host[b]	Source[c]	Geographic origin[d]	FRC member[e]	References
A00102[f]	A^+	Yes	2,152	Maize	PTS	San Joaquin Co., CA	M3120	17,20,26,27
A00149[f]	A^-	Yes	1,458	Maize	PTS	Visalia, CA	M3125	17,20,26,27
A00498	A^-	No	307	Maize-D	JFL	Rossville, KS	M6459	
A00500	A^+	No	2,173	Maize-D	JFL	Rossville, KS	M6460	
A00501	A^+	No	4,425	Maize-D	JFL	Rossville, KS-z	M6461	
A00503	A^-	Yes	2,453	Maize-D	JFL	Rossville, KS-y	M6462	
A00504	A^-	Yes	420	Maize-D	JFL	Rossville, KS-y	M6463	
A00511	A^+	No	2,078	Maize-D	JFL	Rossville, KS-w	M6464	
A00516	A^-	No	1,875	Maize-A	JFL	Rossville, KS-v	M5121	
A00524	A^-	No	419	Maize-D	JFL	Silver Lake, KS-lx	M6465	
A00549	A^-	Yes	2,748	Maize-D	JFL	Silver Lake, KS-2z	M6466	
A00552	A^-	Yes	687	Maize-D	JFL	Silver Lake, KS-2y	M6467	
A00560	A^+	Yes	1,124	Maize-D	JFL	Silver Lake, KS-3	M5122	
A00606	A^-	Yes	2,932	Maize-A	JFL	Cummings, KS	M6468	
A00679	A^+	Yes	1,957	Maize-D	JFL	Highland, KS	M5124	
A00749	A^+	No	435	Sorghum-D	JFL	Powhattan, KS-4	M6469	
A00752	A^+	No	2,750	Sorghum-D	JFL	Powhattan, KS-4z	M5127	
B00278[f]	B^+	Yes	19	Sugarcane	PTS	Taiwan	M3127	26
B00281[f]	B^-	Yes	21	Sugarcane	PTS	Taiwan	M3128	26
D00493	D^+	No	724	Sorghum-A	JFL	St. George, KS	M6470	
D00502[f]	D^+	Yes	964	Maize-D	JFL	Rossville, KS-z	M6471	26
D00506	D^-	No	4	Maize-D	JFL	Rossville, KS-x	M6472	
D00510	D^-	No	1,264	Maize-D	JFL	Rossville, KS-w	M6473	
D00517	D^+	Yes	1,602	Maize-A	JFL	Rossville, KS-v	M6474	
D00522	D^-	Yes	108	Maize-D	JFL	Silver Lake, KS-ly	M6476	
D00523	D^-	Yes	257	Maize-D	JFL	Silver Lake, KS-ly	M6478	
D00525	D^-	Yes	252	Maize-D	JFL	Silver Lake, KS-lx	M6479	
D00526	D^+	Yes	2,002	Maize-D	JFL	Silver Lake, KS-lw	M6480	
D00548	D^+	Yes	884	Maize-D	JFL	Silver Lake, KS-2	M6481	
D00550	D^-	Yes	376	Maize-D	JFL	Silver Lake, KS-2z	M6482	
D00637	D^+	Yes	449	Maize-A	JFL	Troy, KS	M6484	
D00666	D^-	No	46	Maize-D	JFL	Highland, KS	M5123	
D00720	D^-	No	2,618	Sorghum-D	JFL	Powhattan, KS-1	M6485	
D00721	D^-	No	1,146	Sorghum-D	JFL	Powhattan, KS-1	M6486	
D00723	D^-	Yes	95	Sorghum-D	JFL	Powhattan, KS-1	M6487	
D00727	D^+	No	97	Sorghum-D	JFL	Powhattan, KS-1	M6488	
D00737	D^-	Yes	48	Sorghum-D	JFL	Powhattan, KS-2	M6489	
D00738	D^-	Yes	140	Sorghum-D	JFL	Powhattan, KS-2	M6490	
D00741	D^+	Yes	2	Shattercane	JFL	Powhattan, KS-3	M6492	
D00743	D^-	Yes	496	Sorghum-D	JFL	Powhattan, KS-3	M6493	
D00753	D^+	No	85	Sorghum-D	JFL	Powhattan, KS-4z	M6495	
D00875[f]	D^+	Yes	342	Sorghum-D	JFL	Beloit, KS	M5128	26
D02945[f]	D^-	Yes	413	Sorghum	JFL	Holcomb, MS	M3793	26,29
E00434	E^+	No	26	Maize-D	JFL	Breakover, KS	M6496	
E00505	E^-	No	15	Maize-D	JFL	Rossville, KS-x	M6497	
E00507	E^-	Yes	13	Maize-D	JFL	Rossville, KS	M5119	
E00520	E^-	Yes	6	Maize-D	JFL	Silver Lake, KS-lz	M6498	
E00527	E^-	No	8	Maize-D	JFL	Silver Lake, KS-lw	M6499	
E00545	E^-	Yes	146	Maize-A	JFL	Silver Lake, KS-l	M6500	
E00551	E^-	No	16	Maize-D	JFL	Silver Lake, KS-2y	M6501	
E00990[f]	E^-	Yes	...	Maize	JFL	St. Elmo, IL	M3696	26,29
E02192[f]	E^+	Yes	...	Maize	JFL	St. Elmo, IL	M3693	26,29
F00728	F^+	No	2	Sorghum-D	JFL	Powhattan, KS-1	M5598	22,26
F00921	F^-	No	6	Sorghum-D	JFL	Chase, KS	M5132	22
F00965	F^+	No	5	Sorghum-D	JFL	Zeandale, KS	M5134	22
F01051	F^+	No	9	Sorghum-A	JFL	Alma, KS	M5136	22
F01054	F^-	No	5	Sorghum-A	JFL	Overbrook, KS	M5594	22
F01087	F^-	No	3	Sorghum-D	JFL	Centerville, KS	M5595	22
F01106	F^-	No	35	Sorghum-D	JFL	Hallowell, KS	M5138	22
F01137	F^+	No	9	Sorghum-A	JFL	Moline, KS	M5596	22
F01183	F^+	No	3	Sorghum-D	JFL	Moundridge, KS	M5590	22
F01321	F^+	No	3	Sorghum-D	JFL	Enosdale, KS	M5591	22
F01377[f]	F^+	Yes	3	Sorghum-D	JFL	WaKeeney, KS	M5555	22,26
F01518[f]	F^-	Yes	3	Lab cross	JFL	...	M5556	22,26
F04091[f]	F^-	Yes	...	Lab cross	JFL	...	M6561	22
F04092[f]	F^+	Yes	...	Lab cross	JFL	...	M6562	22
F04093[f]	F^-	Yes	...	Lab cross	JFL	...	M6563	22
F04094[f]	F^+	Yes	...	Lab cross	JFL	...	M6564	22

[a] Letter (A–F) indicates the mating population to which a strain belongs; +/− indicates mating type within the mating population.
[b] D = Diseased plant with stalk rot; A = asymptomatic plant.
[c] PTS = Philip T. Spieth, Department of Plant Pathology, University of California, Berkeley; JFL = John F. Leslie, Department of Plant Pathology, Kansas State University, Manhattan.
[d] All isolates from the same location are from the same field unless the location is followed by a number. All isolates are from different plants unless the location is followed by a letter; isolates from the same location with identical letters are from the same plant.
[e] Fusarium Research Center, Department of Plant Pathology, Pennsylvania State University, University Park.
[f] Mating-type standard strain.

a recent survey (29) isolates belonging to *Fusarium* spp. in the *Liseola* section were recovered from all maize and sorghum fields sampled; *F. moniliforme* and *F. proliferatum* predominated. In parallel studies (7,28), members of the A, D, and F mating populations were recovered most frequently, and they collectively accounted for more than 50% of the *Fusarium* isolates recovered from maize and sorghum. Isolates belonging to the B and E populations also were recovered but at a lower frequency. Thus, the members of all of these mating populations are sufficiently widely dispersed to warrant a study of the relative ability of different members of these mating populations to produce fumonisin B_1.

Our objectives in this study were to determine if members of the mating populations of *G. fujikuroi* most commonly associated with maize and sorghum in the United States (A, D, E, and F) were equally capable of producing fumonisin B_1 and to identify sexually fertile strains that could produce differential levels of this toxin for use in studies of the genetic and biochemical bases of toxin synthesis.

MATERIALS AND METHODS

Strains. All strains (Table 1) originated from single uninucleate microconidial cultures. Vegetative cultures were maintained on complete medium (8) and stored at −80 C to minimize genetic changes (58). Genetic nomenclature follows that suggested by Yoder et al (58) for plant-pathogenic fungi except that for mating type designations, which follow those of Leslie (26).

Crossing procedures. Crosses were made on carrot agar (20). Initially, all crosses were made with the standard testers noted in Table 1 as the female parents and the uncharacterized field isolates as the male parents. All crosses to determine mating type were repeated at least twice. Once mating type and mating population were identified, strains were tested for female fertility by reciprocal crosses in which the field isolate functioned as the female parent and the standard tester as the male parent.

Fumonisin B_1 production. The ability of different strains to produce fumonisins was measured following culture on maize grain. Erlenmeyer flasks (300 ml) with Morton closures were filled with 50 g of coarsely cracked maize and 11 ml of distilled water and then autoclaved for 30 min. After the flasks were autoclaved, an additional 11 ml of sterile distilled water was added to each flask. The grain in each flask was inoculated with approximately 10^7 conidia in a water suspension. Flasks were shaken once or twice daily to more evenly distribute the inoculum. Flask cultures were incubated in the dark for 30 days at 25 C.

Fumonisin B_1 assays. Each maize culture was assayed for fumonisins by soaking 5 g of culture material in 100 ml of distilled water for 2–3 h with mixing every half hour. This suspension was filtered through Whatman No. 1 filter paper in a Büchner funnel, and the filtrate then was filtered through a Millipore SC filter with an AP25 prefilter (Millipore Corp., Bedford, MA), collected, and evaporated to dryness at 65 C on a rotary evaporator. The residue was suspended in 10 ml of water, and a 2-ml aliquot (1 g equivalent) was evaporated at 70 C under nitrogen and hydrolyzed in 2 N aqueous KOH. A portion of the hydrolysis product then was derivatized with *N*-methyl-*bis*(trifluoroacetamide) (MBTFA) and analyzed with gas chromatography/mass spectrometry (GC/MS) as described by Plattner et al (42). The sensitivity of this GC/MS method for fumonisin B_1 detection was approximately 1 ppm in maize cultures.

RESULTS

Fumonisin B_1 assays. The variability of fumonisin B_1 levels of replicate cultures grown concurrently was generally ±20–30%, which was approximately the variability of the entire culturing/GC/MS analysis procedure. In most cultures, three fumonisin homologues were detected by the GC/MS procedure. The predominant fumonisin homologue present was fumonisin B_1, which comprised 70–95% of the fumonisins present. Fumonisin B_2 (12) and one of its isomers, fumonisin B_3 (43), constituted the remaining 5–30% of the fumonisins detected. None of the A series of fumonisins was detected in any of the cultures that we assayed. Cultures generally fall within one of three broad classes for fumonisin production: low (< 50 ppm), intermediate (50–500 ppm), and high (> 500 ppm). Within these classes variability can be quite large, e.g., in the low class, strains E00434 (15–37 ppm) and E00520 (2–10 ppm); in the intermediate class, strain D00738 (110–169 ppm); and in the high class, strains A00149 (159–4,832 ppm) and A00102 (1,233–2,900 ppm). Similar variability also has been encountered by other investigators (27,37,45). Culture conditions can affect the actual amount of fumonisins produced, but the relative abilities of different strains to produce fumonisin B_1 were not usually altered by culture conditions. The maize used as a substrate in this study had a low but detectable level of naturally occurring fumonisin B_1 (1–2 ppm).

Mating type and fertility determinations. We examined 56 strains from four different mating populations of *G. fujikuroi* from Kansas for ability to synthesize fumonisin B_1 (Table 1). Only strains (when used as the male) that produced fertile perithecia with oozing ascospores when crossed with standard mating-type testers (used as the female) were included in this study because we wanted to determine if fumonisin B_1 production was related to the mating population to which a strain belonged. Each strain was fertile only with testers of one mating type from only one of the six mating populations.

Strain origin. The strains used in this study were predominantly from diseased (stalk rot) maize and sorghum from 23 fields at 18 sites in Kansas. The exceptions included several strains from asymptomatic plants, one strain from diseased shattercane, and several of the standard mating-type testers that originated outside of the state. Some strains in this study originated in the same field or from the same plant, and genetically distinct (different mating types or different vegetative compatibility groups) strains belonging to the same mating population and strains from different mating populations could be recovered from the same plant.

Fumonisin B_1 levels. Variation in the ability of strains from the same mating population to produce fumonisin B_1 was quite large and is summarized in Table 2. Strains from the A mating population produced the most fumonisin B_1 with an average of 1,786 ppm, members of the D population averaged 636 ppm, members of the E population averaged 33 ppm, and members of the F population averaged 7.5 ppm. Although only two members of the B mating population were tested, the observed levels are similar to those for the E population strains and to those of other strains of *F. subglutinans* that have been described by others (51). The range of levels of toxin produced was quite large in both the A and D populations and considerably less within the E and F populations. In all cases, the standard deviations were large (approximately equal to the mean). The ranges in the amount of toxin produced and the average amount of toxin produced per strain were similar, whether the isolates were re-

TABLE 2. Fumonisin B_1 production (ppm) by isolates in the A, D, E, and F mating populations of *Gibberella fujikuroi* recovered from maize and sorghum in Kansas

Mating population	Maize			Sorghum			Total	
	Average	Range	Number of isolates	Average	Range	Number of isolates	Average	Number of isolates
A	1,815	307–4,425	13	1,593	435–2,750	2	1,786	15
D	684	4–2,002	12	579	48–2,618	10	636	22
E	33	6–146	7	...	...	0	33	7
F	...	...	0	7.5	2–35	11	7.5	11

covered from maize or from sorghum and whether the plant was asymptomatic or diseased.

Among isolates from a single field, variation in the ability to produce fumonisin B_1 could be dramatic. For example, strains A00501 and A00504 are both from maize in Rossville, KS, and yet strain A00501 produced 10 times more fumonisin B_1 than did A00504 (4,425 ppm as compared with 420 ppm). Strains A00503 and A00504 both were recovered from the same plant, but the difference between them in fumonisin B_1 production was quite large (2,453 ppm as compared with 420 ppm). Differences between isolates belonging to the D mating population from the same site were not as extreme, but the range was 4–1,602 ppm with strains D00517 and D00506. Similar differences also were noted with isolates from Powhattan and Silver Lake. The lack of similar variability in the E and F populations suggests that the mating population to which a strain belonged was more important in determining the amount of this toxin that was produced than was the plant host or the geographic origin from which the strain was recovered.

DISCUSSION

Assignment of a strain to a mating population may indicate the potential of that strain to produce fumonisin B_1. For example, the strains in the A mating population all produced significant levels of this mycotoxin while most E and F strains did not. D strains showed the most variation, in a qualitative sense, ranging from background to significant levels. Within a mating population, quantitative variation in fumonisin levels may be quite large, and the distributions observed for the different mating populations overlap substantially. In some cases, large differences in fumonisin B_1 production can be observed among strains recovered from the same field or even from the same plant. As an additional complication, members of more than one mating population can be recovered from a single plant, and genetically distinct individuals in the same mating population can be recovered from the same plant or seed lot (10,21). Thus, studies that rely on a single isolate per plant, feed sample, or other unit may be misleading because the strain that produced the implicated mycotoxin or that was responsible for the observed plant disease might not be recovered from the host material.

Although members of all six mating populations can be recovered from both maize and sorghum (28), the differences in the composition of the pathogen populations recovered from the different hosts could lead to differences in mycotoxin contamination of the host plants as well. The A (50%), D (32%), and E (11%) mating populations predominate in maize, and the D (13%) and F (74%) mating populations predominate in sorghum (29). Based on these distributions, fumonisin B_1-related toxicity problems are probably less likely with sorghum than with maize. This conclusion is consistent with the results of Nelson et al (37), since many of the *F. moniliforme* strains that they recovered from sorghum and millet produced no more than a trace of fumonisin B_1. This conclusion is tempered by several other considerations, however. First, our toxin production data were collected from strains that originated in Kansas, and the geographic base of strains that have been analyzed for both mating population and fumonisin B_1 production levels needs to be broadened. Second, more data are available on fumonisin B_1 from maize or maize-associated strains than are available from sorghum or sorghum-associated strains. Risks to other crops that are known to be infected by this fungus, such as rice, asparagus, mangoes, sugarcane, and pineapple, remain essentially unknown. Third, the ability of these strains to produce fumonisins on substrates other than maize seed, e.g., maize stalks and sorghum grain and stalks, has not been examined. Fourth, most of the strains used in this study originated from stalks rather than from grains or grain-based feedstuffs. The composition of *G. fujikuroi* populations in maize seed (10) with respect to mating population is somewhat different from that found in maize or sorghum stalks (29), so our samples may be different from those that would be recovered from grains or grain-based feeds. Furthermore, none of the strains examined in this study is from sources that are known to be associated with equine leukoencephalomalacia or other fumonisin B_1-related disorders. Thus, we feel that our data more accurately reflect the distribution of toxin-producing strains in field populations than do studies that are limited primarily to strains recovered from feedstuffs associated with one of these disorders.

Further work also is warranted on the basic biology of *G. fujikuroi* and its ability to synthesize fumonisin B_1 because some significant questions remain unanswered. 1) The degree to which fumonisin-producing strains are related is unknown. Twenty-five toxin-producing strains were not clones of one another (27), but no correlated characteristic(s) for distinguishing high fumonisin-producing strains from strains that produce lower levels of these compounds are presently available. For example, molecular techniques might be used to identify a suitable marker, perhaps based on restriction fragment length polymorphism (RFLP) or rapid analysis of polymorphic DNA (RAPD) methodologies, that would permit rapid, accurate identification of strains that produce high levels of this toxin. 2) The taxonomic status of the different mating populations needs to be more clearly defined (members of both A and F mating populations are *F. moniliforme* using present criteria). Morphological characters that differentiate these groups at the species level need to be identified to reduce or eliminate the problems that result from having several different mating populations (= biological species) labeled with the same name. 3) Environmental conditions that affect the production of this toxin need further study, and methodologies should be developed to reduce the amount of fumonisins present in commercial products. 4) Competitive nontoxigenic strains might be useful in a biological control-type program that would reduce the number of toxin-producing fungal propagules below a threshold that is necessary for toxin-associated disorders to be manifested. 5) The genetic and biochemical bases of fumonisin biosynthesis and its regulation need to be elucidated to determine whether biosynthesis of this compound is regulated in a similar manner in the different mating populations.

In conclusion, many strains within *Fusarium* section *Liseola* apparently are capable of making significant quantities of fumonisin B_1, and these strains are not limited to diseased plant material. The work reported here will further genetic and biochemical studies of this class of mycotoxins and may provide a starting point for chemotaxonomy within the *Liseola* section such as has been used in other *Fusarium* sections (36).

LITERATURE CITED

1. Badiali, L., Mamdouh, H., Abou-Youssef, H., Radwan, A. I., Hamdy, F. M., and Hildebrandt, P. K. 1968. Moldy corn poisoning as the major cause of an encephalomalacia syndrome in Egyptian equidae. Am. J. Vet. Res. 29:2029-2035.
2. Biester, H. E., and Schwarte, L. H. 1939. Moldy corn poisoning (leukoencephalomalacia) in horses with history of previous attack as well as recovery from virus encephalomyelitis. N. Am. Vet. 20(6):17-19.
3. Biester, H. E., Schwarte, L. H., and Reedy, C. H. 1940. Further studies on moldy corn poisoning (leukoencephalomalacia) in horses. Vet. Med. 35:636-639.
4. Brownie, C. F., and Cullen, J. 1986. Characterization of experimentally-induced equine leukoencephalomalacia (ELEM) in ponies (*Equus caballus*): A preliminary report. Vet. Hum. Toxicol. 29:34-37.
5. Buck, W. B., Haliburton, J. C., Thilsted, J. P., Lock, T. F., and Vesonder, B. S. 1979. Equine leukoencephalomalacia: Comparative pathology of naturally occurring and experimental cases. Annu. Proc. Am. Assoc. Vet. Lab. Diagn., 22nd, pp. 239-258.
6. Butler, T. 1902. Notes on a feeding experiment to produce leucoencephalitis in a horse, with positive results. Agric. Vet. Rev. 26:748-751.
7. Chaisrisook, C., and Leslie, J. F. 1990. Genetic diversity within populations of *Fusarium* section *Liseola* from corn and sorghum in Kansas. (Abstr.) Phytopathology 80:1042.
8. Correll, J. C., Klittich, C. J. R., and Leslie, J. F. 1987. Nitrate nonutilizing mutants of *Fusarium oxysporum* and their use in vegetative compatibility tests. Phytopathology 77:1640-1646.
9. Dwinell, L. D., Kuhlman, E. G., and Blakeslee, G. M. 1981. Pitch

canker of southern pines. Pages 188-194 in: *Fusarium:* Diseases, Biology and Taxonomy. P. E. Nelson, T. A. Toussoun, and R. J. Cook, eds. Pennsylvania State University Press, University Park.
10. Farrokhi-Nejad, R., and Leslie, J. F. 1990. Vegetative compatibility group diversity within populations of *Fusarium moniliforme* isolated from corn seed. (Abstr.) Phytopathology 80:1043.
11. Foley, D. C. 1962. Systemic infection of corn by *Fusarium moniliforme.* Phytopathology 52:870-872.
12. Gelderblom, W. C. A., Jaskiewicz, K., Marasas, W. F. O., Thiel, P. G., Horak, R. M., Vleggaar, R., and Kriek, N. P. J. 1988. Fumonisins: Novel mycotoxins with cancer-promoting activity associated with *Fusarium moniliforme.* Appl. Environ. Microbiol. 54:1806-1811.
13. Harrison, L. R., Colvin, B. M., Greene, J. T., Newman, L. E., and Cole, J. R., Jr. 1990. Pulmonary edema and hydrothorax in swine produced by fumonisin B_1, a toxic metabolite of *Fusarium moniliforme.* J. Vet. Diagn. Invest. 2:217-221.
14. Haschek, W. M., and Haliburton, J. C. 1986. *Fusarium moniliforme* and zearalenone toxicoses in domestic animals: A review. Pages 213-235 in: Diagnosis of Mycotoxicoses. J. L. Richard and J. R. Thurston, eds. Martinus Nijhoff Publishers, Boston.
15. Howerth, E. W., Wyatt, R. D., and Hayes, D. A. 1989. Leukoencephalomalacia in a white-tailed deer from North Carolina. J. Wildl. Dis. 25:384-387.
16. Hsieh, W. H., Smith, S. N., and Snyder, W. C. 1977. Mating groups in *Fusarium moniliforme.* Phytopathology 67:1041-1043.
17. Kathariou, S. 1981. Gene pool organization in *Fusarium moniliforme.* Ph.D. dissertation. University of California, Berkeley.
18. Kellerman, T. S., Marasas, W. F. O., Pienaar, J. G., and Naudé, T. W. 1972. A mycotoxicosis of equidae caused by *Fusarium moniliforme* Sheldon. A preliminary communication. Onderstepoort J. Vet. Res. 39:205-208.
19. Kellerman, T. S., Marasas, W. F. O., Thiel, P. G., Gelderblom, W. C. A., Cawood, M., and Coetzer, J. A. W. 1990. Leukoencephalomalacia in two horses induced by oral dosing of fumonisin B_1. Onderstepoort J. Vet. Res. 57:269-275.
20. Klittich, C. J. R., and Leslie, J. F. 1988. Nitrate reduction mutants of *Fusarium moniliforme* (*Gibberella fujikuroi*). Genetics 118:417-423.
21. Klittich, C. J. R., and Leslie, J. F. 1988. Unusual isolates of *Fusarium moniliforme* from sorghum in Kansas. (Abstr.) Phytopathology 78:1519.
22. Klittich, C. J. R., and Leslie, J. F. Identification of a second mating population within *Gibberella fujikuroi* (*Fusarium moniliforme*). Mycologia. In press.
23. Kriek, N. P. J., Kellerman, T. S., and Marasas, W. F. O. 1981. Comparative study of the toxicity of *Fusarium verticillioides* (= *F. moniliforme*) to horses, primates, pigs, sheep, and rats. Onderstepoort J. Vet. Res. 43:129-131.
24. Kuhlman, E. G. 1982. Varieties of *Gibberella fujikuroi* with anamorphs in *Fusarium* section *Liseola.* Mycologia 74:759-768.
25. LaMondia, J. A., and Elmer, W. H. 1989. Pathogenicity and vegetative compatibility among isolates of *Fusarium oxysporum* and *F. moniliforme* colonizing asparagus tissues. Can. J. Bot. 67:2420-2424.
26. Leslie, J. F. 1991. Mating populations in *Gibberella fujikuroi* (*Fusarium* section *Liseola*). Phytopathology 81:1058-1060.
27. Leslie, J. F., Doe, F. J., Plattner, R. D., Shackelford, D. D., and Jonz, J. Fumonisin B_1 production and vegetative compatibility of strains from *Gibberella fujikuroi* mating population "A" (*Fusarium moniliforme*). Mycopathologia. In press.
28. Leslie, J. F., Klittich, C. J. R., and Chaisrisook, C. 1990. Fertility of isolates from *Fusarium* section *Liseola.* (Abstr.) Mycol. Soc. Am. Newsl. 41(1):24.
29. Leslie, J. F., Pearson, C. A. S., Nelson, P. E., and Toussoun, T. A. 1990. *Fusarium* spp. from corn, sorghum, and soybean fields in the central and eastern United States. Phytopathology 80:343-350.
30. Marasas, W. F. O., Kellerman, T. S., Gelderblom, W. C. A., Coetzer, J. A. W., Thiel, P. G., and van der Lugt, J. J. 1988. Leukoencephalomalacia in a horse induced by fumonisin B_1 isolated from *Fusarium moniliforme.* Onderstepoort J. Vet. Res. 55:197-203.
31. Marasas, W. F. O., Kellerman, T. S., Pienaar, J. G., and Naudé, T. W. 1976. Leukoencephalomalacia: A mycotoxicosis of equidae caused by *Fusarium moniliforme* Sheldon. Onderstepoort J. Vet. Res. 43:113-122.
32. Marasas, W. F. O., Kriek, N. P. J., Wiggens, V. M., Steyn, P. S., Towers, D. K., and Hastie, T. J. 1979. Incidence, geographic distribution, and toxigenicity of *Fusarium* species in South African corn. Phytopathology 69:1181-1185.
33. Marasas, W. F. O., and Nelson, P. E., eds. 1987. Mycotoxicology: Introduction to the Mycology, Plant Pathology, Chemistry, Toxicology, and Pathology of Naturally Occurring Mycotoxicoses in Animal and Man. Pennsylvania State University Press, University Park.
34. Marasas, W. F. O., Nelson, P. E., and Toussoun, T. A. 1984. Toxigenic Fusarium Species: Identity and Mycotoxicology. Pennsylvania State University Press, University Park.
35. Martin, J. P., Handojo, H., and Wismer, C. A. 1989. Pokkah boeng. Pages 157-168 in: Diseases of Sugarcane: Major Diseases. C. Ricaud, B. T. Egan, A. G. Gillaspie, Jr., and C. G. Hughes, eds. Elsevier, New York.
36. Miller, J. D., Greenhalgh, R., Wang, Y. Z., and Lu, M. 1991. Trichothecene chemotypes of *Fusarium* species. Mycologia 83:121-130.
37. Nelson, P. E., Plattner, R. D., Shackelford, D. D., and Desjardins, A. E. 1991. Production of fumonisins by strains of *Fusarium moniliforme* from various substrates and geographic areas. Appl. Environ. Microbiol. 57:2410-2412.
38. Nelson, P. E., Toussoun, T. A., and Marasas, W. F. O. 1983. *Fusarium* Species: An Illustrated Manual for Identification. Pennsylvania State University Press, University Park.
39. Nirenberg, H. I. 1989. Identification of *Fusaria* occurring in Europe on cereals and potatoes. Pages 179-193 in: *Fusarium:* Mycotoxins, Taxonomy and Pathogenicity. J. Chelkowski, ed. Elsevier, New York.
40. Phinney, B. O., and West, C. A. 1960. Gibberellins as native plant growth regulators. Annu. Rev. Plant Physiol. 11:411-436.
41. Pienaar, H. G., Kellerman, T. S., and Marasas, W. F. O. 1981. Field outbreaks of leukoencephalomalacia in horses consuming maize infected by *Fusarium verticillioides* (= *F. moniliforme*) in South Africa. J. S. Afr. Vet. Assoc. 52:21-24.
42. Plattner, R. D., Norred, W. P., Bacon, C. W., Voss, K. A., Peterson, R., Shackelford, D. D., and Weisleder, D. 1990. A method of detection of fumonisins in corn samples associated with field cases of equine leukoencephalomalacia. Mycologia 82:698-702.
43. Plattner, R. D., Weisleder, D., Shackelford, D. D., Peterson, R. E., and Powell, R. G. A new fumonisin from solid cultures of *Fusarium moniliforme.* Mycopathologia. In press.
44. Rohrbach, K. G., and Pfeiffer, J. B. 1976. Susceptibility of pineapple cultivars to fruit diseases incited by *Penicillium funiculosum* and *Fusarium moniliforme.* Phytopathology 66:1386-1390.
45. Ross, P. F., Nelson, P. E., Richard, J. L., Osweiler, G. D., Rice, L. G., Plattner, R. D., and Wilson, T. M. 1990. Production of fumonisins by *Fusarium moniliforme* and *Fusarium proliferatum* associated with equine leukoencephalomalacia and a pulmonary edema syndrome in swine. Appl. Environ. Microbiol. 56:3225-3226.
46. Ross, P. F., Rice, L. G., Plattner, R. D., Osweiler, G. D., Wilson, T. M., Owens, D. L., Nelson, H. A., and Richard, J. L. 1991. Concentrations of fumonisin B_1 in feeds associated with animal health problems. Mycopathologia 114:129-135.
47. Schwarte, L. H., Biester, H. E., and Murray, C. 1937. A disease of horses caused by feeding moldy corn. J. Am. Vet. Assoc. 90:76-85.
48. Snyder, W. C., and Hansen, H. N. 1945. The species concept in *Fusarium* with reference to *Discolor* and other sections. Am. J. Bot. 32:657-666.
49. Snyder, W. C., and Toussoun, T. A. 1965. Current status of taxonomy in *Fusarium* species and their perfect stages. Phytopathology 55:833-837.
50. Sun, S.-K., and Snyder, W. C. 1981. The bakane disease of the rice plant. Pages 104-113 in: Fusarium: Diseases, Biology and Taxonomy. P. E. Nelson, T. A. Toussoun, and R. J. Cook, eds. Pennsylvania State University Press, University Park.
51. Thiel, P. G., Marasas, W. F. O., Sydenham, E. W., Shephard, G. S., Gelderblom, W. C. A., and Nieuwenhuis, J. J. 1991. Survey of fumonisin production by *Fusarium* species. Appl. Environ. Microbiol. 57:1089-1093.
52. Thiel, P. G., Shephard, G. S., Sydenham, E. W., Marasas, W. F. O., Nelson, P. E., and Wilson, T. M. 1991. Levels of fumonisin B_1 and B_2 in feeds associated with confirmed cases of equine leukoencephalomalacia. J. Agric. Food Chem. 39:109-111.
53. Thomas, M. D., and Buddenhagen, I. W. 1980. Incidence and persistence of *Fusarium moniliforme* in symptomless maize kernels and seedlings in Nigeria. Mycologia 72:882-887.
54. Varma, A., Lele, V. C., Raychaudhuri, S. P., Ram, A., and Sang, A. 1974. Mango malformation: A fungal disease. Phytopathol. Z. 79:254-257.
55. Wiebe, L. A., and Bjeldanes, L. F. 1981. Fusarin C, a mutagen from *Fusarium moniliforme* grown on corn. J. Food Sci. 46:1424-1426.
56. Wilson, T. M., Nelson, P. E., Marasas, W. F. O., Thiel, P. G., Shephard, G. S., Sydenham, E. W., Nelson, H. A., and Ross, P. F. 1990. A mycological evaluation of feed from 41 farms with equine leukoencephalomalacia. J. Vet. Diagn. Invest. 2:352-354.
57. Wilson, T. M., Ross, P. F., Rice, L. G., Osweiler, G. D., Nelson, H. A., Owens, D. L., Plattner, R. D., Reggiardo, C., Noon, T. H., Pickrell, J. W. 1990. Fumonisin B_1 levels associated with an epizootic of equine leukoencephalomalacia. J. Vet. Diagn. Invest. 2:213-216.
58. Yoder, O. C., Valent, B., and Chumley, F. 1986. Genetic nomenclature and practice for plant pathogenic fungi. Phytopathology 76:383-385.