Actinobacillus suis infection in pigs in southwestern Ontario

S. Ernest Sanford, Gaylan K.A. Josephson, Abdul J. Rehmtulla, Anne M.E. Tilker

Abstract

Actinobacillus suis **was isolated from tissues of 39 pigs, 2 porcine lungs, and 1 uterine swab submitted for diagnostic evaluation from 24 farms in southwestern Ontario between 1985 and 1988. These isolates represented a gradually increasing incidence of herd outbreaks caused by *A. suis* in southwestern Ontario. The outbreaks were typified by sudden death in suckling or recently weaned pigs; 87% of the affected pigs examined at the laboratory were between two and 28 days old. Petechial to ecchymotic hemorrhages in the thoracic and abdominal organs accompanied by serofibrinous exudates in both cavities were the most common gross lesions. The lesions were characterized histologically by bacterial thromboembolism and necrosis randomly scattered in thoracic and abdominal organs. Occasionally, bacterial thromboemboli were surrounded by centrifugally radiating, eosinophilic, club-like colonies. Diffuse necrohemorrhagic myocarditis that was more severe in the atria, and diffuse subacute meningoencephalitis, were less frequent but distinctive lesions. Multiple litters were affected in most herd outbreaks, and mortality often approached 50% in affected litters. Although the *A. suis* organism was susceptible to nearly every antibiotic against which it was tested, the suddenness of herd outbreaks precluded attempts at treatment.**

Résumé

Infections par *Actinobacillus suis* chez le porc dans le Sud-Ouest ontarien

Actinobacillus suis **fut isolé de tissus provenant de 39 porcs, de poumons de deux porcs et d'un écouvillon utérin soumis pour fins de diagnostic par 24 fermes du Sud-Ouest ontarien entre 1985 et 1988. Ces résultats indiquent une incidence accrue de cas d'actinobacillose dans le Sud-Ouest ontarien. Les épidémies se caractérisaient par de la mortalité subite chez les porcelets à la mamelle ou chez ceux qui venaient tout juste d'être sevrés; 87 % des porcs examinés au laboratoire avaient entre 2 et 28 jours. Si les porcelets les plus jeunes succombaient, l'éleveur avait tendance à assumer initialement qu'ils avaient été tout simplement écrasés par la truie. Les lésions macroscopiques les plus communément rencontrées furent des pétéchies et des ecchymoses des organes abdominaux et thoraciques avec des épanchements exsudatifs sérofibrineux dans les deux cavités. À l'examen microscopique, ces dernières se caractérisaient par des lésions de nécrose et de thrombo-embolisme bactérien réparties au hasard dans les organes thoraciques et abdominaux. À l'occasion, les thrombo-embolismes bactériens furent entourés de colonies éosinophiliques, en forme de massues et irradiant de façon centrifuge. Une myocardite nécro-hémorragique, plus sévère dans les oreillettes, et une méningo-encéphalite subaiguë diffuse représentaient d'autres lésions destructives mais moins fréquemment rencontrées. Plusieurs portées furent affectées dans un même élevage et le taux de mortalité approchait 50 %. La rapidité avec laquelle la maladie s'est déclarée dans les élevages a fait en sorte que les traitements appropriés n'ont pu être institués bien que *A. suis* soit sensible à presque tous les antibiotiques contre lesquels il fut évalué.**

Can Vet J 1990; 31: 443-447

Veterinary Laboratory Services, Ontario Ministry of Agriculture and Food, Huron Park, Ontario N0M 1Y0.

Introduction

Even though *Actinobacillus suis* infections in pigs have occasionally been reported from diverse locales around the world (1-6), this disease remains obscure. These sporadic reports have usually described septicemia and death in suckling or recently weaned pigs.

In this paper, we report an annually increasing incidence of *A. suis* infection causing primarily septicemia and sudden death in suckling pigs in southwestern Ontario.

Materials and methods

This study is based on laboratory findings from the examination of 39 porcine carcasses, 2 lungs, and 1 uterine swab, submitted to the Huron Park Diagnostic Laboratory (HPDL), from which *A. suis* was isolated between January 1985 and December 1988. In this four-year period, 4819 porcine carcasses were submitted to the HPDL for evaluation: 1239 of these in 1985, 1055 in 1986, 1284 in 1987, and 1241 in 1988. From 1978 to 1988 inclusive, an average of 1446 porcine carcasses with a range of 1055 (in 1986) to 1799 (in 1979) were submitted annually to the laboratory. All pigs in this study came from herds which had modern intensive management systems in which large numbers of pigs are kept confined indoors, and isolated from other herds. Histories and clinical signs were obtained by interviewing producers and/or their veterinarians when carcasses or tissues were submitted. All pigs were necropsied routinely. Portions of organs selected for bacteriological culture were streaked on 5% bovine blood agar and incubated at 37°C; plates were examined at 24 and 48 hours postinoculation. Identification of *A. suis* was based on previously established characteristics (1,4,5) and has remained unchanged at the HPDL for more than a decade. The principal features of differentiation from other bacteria isolated from pigs include: gram-negative, non-mucoid, coccobacillary and sometimes filamentous

Table 1. Isolations of *Actinobacillus suis*, 1985–1988

Case no.	Clinical characteristics	Submitted material	Age	Organ
1985				
1	vaginal discharge	1 uterine swab	2 yr	uterus
1986				
2	sudden death	2 piglets	21 d	multiple
3	sudden death	6 piglets	10 d	multiple
4	sudden death	1 piglet	2 d	multiple
1987				
5	pneumonia	1 lung	5 mo	lung
6	sudden death	2 piglets	17 d	multiple
7	diarrhea	1 piglet	11 d	multiple
8	nervous signs	1 piglet	10 wk	meninges
9	lameness	3 piglets	28 d	joints, serosae
10	sudden death	1 piglet	12 d	multiple
11	nervous signs	1 piglet	10 d	meninges
1988				
12	red skin lesions	1 sow	2 yr	multiple
13	sudden death	1 piglet	21 d	multiple
14	sudden death	2 piglets	20 d	multiple
15	sudden death	1 piglet	17 d	multiple
16	sudden death	1 piglet	3 d	multiple
17	diarrhea	1 piglet	12 d	tonsil, intestine
18	coughing	1 piglet	49 d	tonsil
19	sudden death	2 piglets	7 d	multiple
20	pneumonia	1 piglet	28 d	multiple
21	sudden death	1 piglet	14 d	multiple
22	diarrhea	2 piglets	10 d	intestines
23	diarrhea	2 piglets	9 d	intestines
24	greasy skin	2 piglets	14 d	subcutaneous lymph node
25	diarrhea	1 piglet	49 d	tonsil
26	sudden death	1 piglet	14 d	multiple
27	pneumonia	1 lung	84 d	lung
28	anorexia	1 boar	10 mo	lung, tonsil

Note: Pigs from cases 2 and 9; 12 and 13; 14 and 27; and 19 and 20, respectively, were from the same farm

structure; nonmotile; growth on MacConkey agar; complete hemolysis on bovine blood agar; catalase, oxidase, and urease production; hydrolysis of esculin; acid production from salicin and trehalose, without gas, but no production from mannitol. The antimicrobial susceptibility of all *A. suis* isolates was determined by disk-diffusion tests on blood-supplemented Mueller-Hinton agar (7) against ampicillin, carbenicillin, cephalothin, gentamicin, kanamycin, neomycin, penicillin G, polymyxin B, streptomycin, sulfisoxazole, tetracycline, and trimethoprim-sulfamethoxazole. Tissue samples for histological examination were fixed in 10% neutral buffered formalin, sectioned at 6 μm, and stained with hematoxylin and eosin.

Results

From 1985 to 1988 inclusive, *A. suis* was isolated, usually as the predominant or only organism, from tissues of 39 pigs, 2 lungs, and 1 uterine swab representing 28 separate submissions from 24 different farms (Table 1). The number of isolations of *A. suis* more than doubled each year during the study period, even though the number of submissions remained about the same. Infected pigs ranged in age from two days to two years old but, typically, a herd outbreak was characterized by sudden death in suckling pigs; 34 of the 39 (87%) pigs examined from 18 of 24 farms were between 2 and 28 days old. Only one litter was affected in each of three herds (cases 15, 16, and 21), whereas multiple litters were involved in most outbreaks (cases 2–4, 6, 10, 11, 14, 19, and 20). Mortality often approached 50% in affected litters. Occasionally, *A. suis* was isolated from intestines and mesenteric lymph nodes of scouring pigs. In all of these pigs however, some other agent was identified as the cause of, or the major contributor to, the diarrhea.

The first *A. suis* isolate was from a uterine swab taken from a nonpregnant sow with a vaginal discharge (Table 1). That was the only *A. suis* isolate from a reproductive tract during the study period, but the number of porcine reproductive tracts cultured annually (<10) at this laboratory is small. Toward the end of the observation period, isolates of *A. suis* were sometimes made from tonsils and/or lungs (cases 17, 18, 25, and 28), and once (case 24) from a subcutaneous lymph node from a pig with exudative epidermitis, and seemed to be unrelated to any disease process in the pigs from which they were isolated.

Clinical signs

The most common clinical history was sudden death of several pigs in one or two litters in a farrowing room

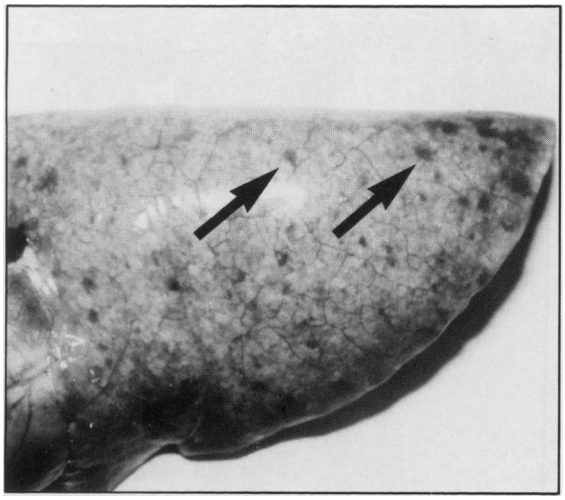


Figure 1. Petechial hemorrhages and necrosis (arrows) in the lung of a two-week-old pig with *Actinobacillus suis* septicemia.

as was reported in cases 2–4, 13, 15, 16, 19, 20, 21, and 26 (Table 1). In two of these herds, the owners had initially thought that the dead pigs had been crushed by the sow, but became concerned that too many pigs had been found dead. This caused them to submit pigs to the laboratory. When clinical signs were noted in affected sucklings, they consisted of short periods of panting accompanied by shaking and/or paddling (cases 6, 11, and 14). Cyanosis and 2–3 mm purple spots randomly scattered over the body surface of all male pigs from two litters which had been castrated 24 h previously were the presenting signs in case 10. Cyanosis or reddening of the skin was also observed in the pigs submitted in outbreaks 13–15. Central nervous system (CNS) dysfunction characterized by ataxia and stumbling was seen in the 10-week-old pig submitted in outbreak 8. Most herds were affected only once, or lost pigs over a short period of a few days. The outbreak then subsided as suddenly as it had begun. In three herds, however, second and third outbreaks (almost identical to the first) occurred several weeks or months later. The findings in case 12 have been published as part of a paper on *A. suis* infections affecting mainly sows and resembling erysipelas outbreaks (8).

Gross and histopathological findings

The most striking gross lesions were petechial to ecchymotic hemorrhages in one or more of the following organs: lung, heart, liver, kidney, spleen, skin, and intestines. These were seen in pigs in cases 2, 3, 6, 10, 14–16, and 19–21 (Figures 1 and 2). Increased serous or serofibrinous exudates in the thoracic and abdominal cavities accompanied the hemorrhages in most cases but were particularly noticeable in cases 3, 6, 14, and 19–21, and were the significant findings, without hemorrhage, in case 9. Hemopericardium accentuated the fibrinohemorrhagic myocardial lesions in cases 6, 14, 15, and 19. There were increased amounts of cerebrospinal fluid and the meninges were cloudy in cases 8 and 11.

Bacterial thromboembolism with accompanying fibrinohemorrhagic necrosis in randomly scattered foci

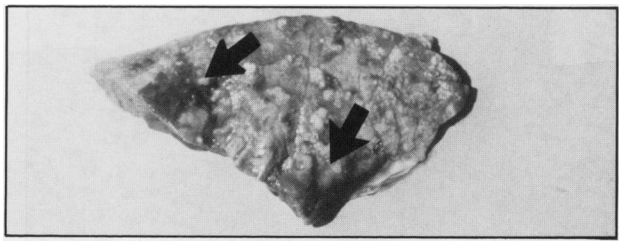


Figure 2. Large coalescing areas of pulmonary hemorrhage and necrosis (arrows) in a two-day-old pig with *A. suis* septicemia. Lesions occurred in all lobes.

in various organs including lung, liver, kidney, spleen, heart, meninges, and brain was the distinctive histological finding (Figures 3 and 4). The lung was most often involved, and the most severely affected lungs exhibited numerous irregular, randomly scattered, often coalescing areas of necrosis surrounding bacterial colonies. These bacterial colonies were sometimes surrounded by amorphous, centrifugally radiating, eosinophilic material mixed with necrotic debris (Figure 4). In less severely affected lungs, the changes were characterized by subpleural, interlobular, and intra-alveolar edema, interstitial pneumonia, serofibrinous pleuritis, and various numbers of bacterial thromboemboli (Figure 3). Bacterial thromboemboli in the center of necrotic foci, with and without radiating, eosinophilic club-like colonies, were scattered randomly but less common in liver, kidney, spleen, and myocardium than in lung. A fulminant, diffuse, necrohemorrhagic myocarditis, more severe in the atria, typified the microscopic changes in cases 6, 14, 15, and 19. Marked diffuse subacute meningoencephalitis and bilateral subacute Gasserian ganglioneuritis characterized the changes in pigs with CNS lesions. Meningoencephalitis consisted of an influx of large numbers of predominantly mononuclear inflammatory cells into the leptomeninges, perivascularly in the cerebral and cerebellar cortices, filling the lateral ventricles and invading the subventricular neuropil. Bacterial thromboembolism in subcutaneous vessels, accompanied by full-thickness necrosis of the skin and subtended by a band of a mixture of degenerating inflammatory cells, occurred in cases 12 and 13.

Antimicrobial susceptibility findings

All 28 *A. suis* isolates were susceptible to ampicillin, carbenicillin, gentamicin, kanamycin, penicillin G, polymyxin B, and trimethoprim-sulfamethoxazole. One isolate was resistant to tetracycline and sulfisoxazole, and had an intermediate susceptibility to streptomycin. Another isolate was resistant to tetracycline only.

Discussion

Septicemia causing rapid death in suckling and weanling pigs up to 12 weeks old has been the predominant feature in previous reports of *A. suis* disease in pigs (1–6). Similarly, 87% of the pigs in this study were between 2 and 28 days of age, still suckling, and most had experienced septicemia causing sudden death. Meningitis and arthritis, though not unexpected in septicemia, have been less frequently reported than

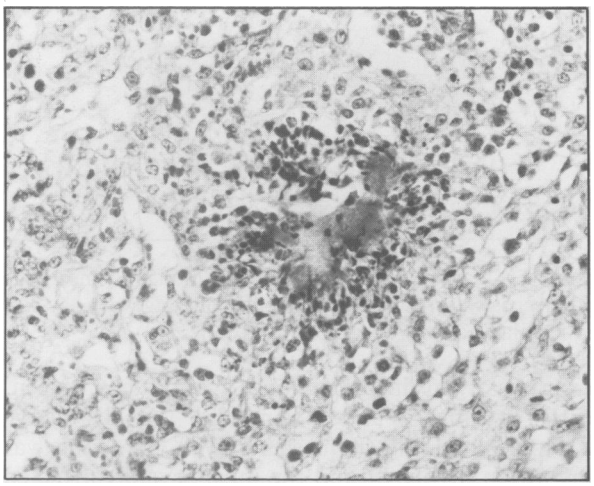


Figure 3. Histological lesions in lung shown in Figure 1; interstitial pneumonia and a necrotic focus filled with *A. suis* bacterial in club colony formation.

septicemia, and were features in only a few of our cases. It would seem that, although the *A. suis* organism causes severe septicemia including polyserositis in young pigs, it has a great affinity for the parenchyma of the internal organs. Certainly, in the outbreaks reported here, the parenchyma of the various organs, via embolic spread, were more frequent targets than the serous membranes.

When it occurred, myocarditis was a striking feature in pigs in this study. Although myocarditis had been documented in previous reports on *A. suis* outbreaks, it did not seem to have been a significant feature of those outbreaks (4,5). Similar to previous reports (4,9), the significance of *A. suis* infection in the reproductive tract of the sow in our study remained obscure.

Although the pathogenesis has not been fully explored, *A. suis* seems to affect primarily young pigs, especially neonates, up to and just beyond weaning age. It probably gains entry into the pig either through the mucous membranes or via an abrasion, then spreads systemically. Researchers have reproduced disease in pigs by intravenous injections of *A. suis* organisms (3). From their work, Mair *et al* suggested that very young pigs were more susceptible to experimental infection with *A. suis* than older pigs (4). Of six, 13-day-old piglets inoculated intraperitoneally, all became febrile in 24 hours, two died of generalized disease within three days, and the four survivors all had chronic pleuritis and peritonitis when necropsied 10 days postinoculation (4).

The *A. suis* organism was susceptible to nearly every antibiotic that it was tested against, including tetracycline. Because of the acute nature of most outbreaks, however, there had not been time to attempt treatment. If these outbreaks could be anticipated, antibiotic treatment would likely be effective.

Reviewing earlier reports of *A. suis* outbreaks, it appears that these outbreaks occurred sporadically in various locales, and circulated among a few herds for periods varying from months up to a year or two. Then, if the lack of further reports from the various countries is any indication, the outbreaks stopped as suddenly as they had started (1–6). Prior to 1985, the *A. suis* organism had never been isolated from specimens submitted to our laboratory. The cultural protocol for isolation of *A. suis* has remained unchanged for over a decade. Yet, as shown in this study (Table 1), there has been an annual increase between 1985 and 1988 in the number of *A. suis* isolations made at this laboratory, primarily from suckling pigs which had died suddenly on different farms in southwestern Ontario.

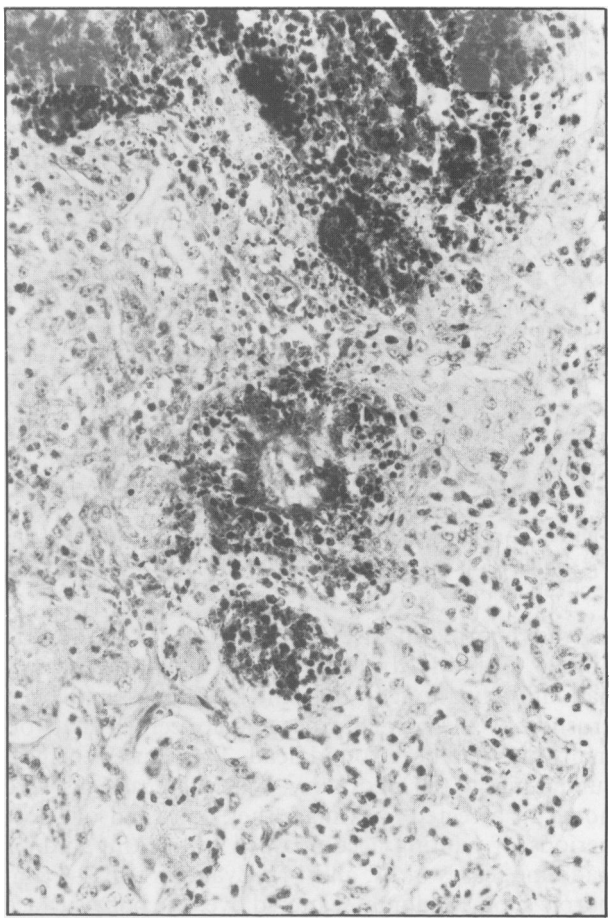


Figure 4. Severe fibrinonecrotizing pneumonia in the same lung as shown in Figure 2. Note the coalescing areas of necrosis filled with fibrinocellular exudate.

It would seem that the *A. suis* organism emerges as a pathogen for a short time in a new geographical area, probably where there has been some subtle change in susceptibility of the pig population. This change might be a shift to more minimal disease or SPF herds or more herds with other types of high health status. It then acts as an opportunistic pathogen among neonates on those farms and soon reequilibrates in the upgraded swine population as a commensal rarely causing disease outbreaks. It is reasonable then to expect that the number of annual outbreaks here will soon peak. Indeed, for the first time since the initial isolation of *A. suis* in 1985, there was a decrease in the number of *A. suis* isolations at this laboratory. In 1989, *A. suis* was isolated from 12 pigs representing 10 separate submissions from eight different farms. The isolates represented new cases in six of the eight herds. Although the *A. suis* outbreaks may now have peaked in Ontario, veterinarians and producers should be on the alert since similar outbreaks are likely to occur in other pig-producing areas in the near future. CVJ

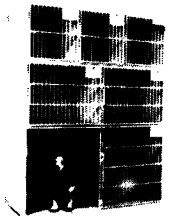
References

1. VanDorssen CA, Jaartsveld FHJ. *Actinobacillus suis* (*novo species*), a bacterium occurring in swine. Tijdschr Diergeneeskd 1962; 87: 450–458.
2. Zimmermann T. Die actinobazillose des schweines. Tieraerztl Umsch 1965; 12: 565–567.
3. Cutlip RC, Amtower WC, Zinober MR. Septic embolic actinobacillosis of swine: A case report and laboratory reproduction of the disease. Am J Vet Res 1972; 33: 1621–1626.
4. Mair NS, Randall CJ, Thomas GW, Harbourne JF, McCrea CT, Cowl KP. *Actinobacillus suis* infection in pigs. J Comp Pathol 1974; 84: 113–119.
5. MacDonald DW, Hewitt MP, Wilton GS, Rawluk S, Childs L. *Actinobacillus suis* infections in Alberta swine, 1973–75: Pathology and bacteriology. Can Vet J 1976; 17: 251–254.
6. Liven E, Larsen HJ, Lium B. Infection with *Actinobacillus suis* in pigs. Acta Vet Scand 1978; 19: 313–315.
7. Barry AL, Thornsberry C. Susceptiblity tests: Diffusion test procedures. In: Lennette EH, Balows A, Hausler WJ, Shadomy HJ, eds. Manual of Clinical Microbiology. 4th ed. Washington, D.C: American Society for Microbiology, 1985: 978–987.
8. Miniats OP, Spinato MT, Sanford SE. *Actinobacillus suis* septicemia in mature swine. Two outbreaks resembling erysipelas. Can Vet J 1989; 30: 943–947.
9. Ross RF, Hall JE, Orning AP, Dale SE. Characterization of an *Actinobacillus* isolated from the sow vagina. Int J Syst Bacteriol 1972; 22: 39–46.

Need Cages?
Save 33 1/3%

Clark Cages
"Committed to Excellence"

- Quiet & Warm
- Sensibly Priced
- Durable
- Attractive
- Easy to Assemble



Clark Cage Dryer

- Heavy - duty construction
- Fully - assembled
- Virtually maintenance - free
- Dries animal quickly and safely
- Space - saver design

For a *free* colour brochure and price list contact:

Clark Cages Inc.
Canada (705) 474-8303
USA Texas - (214) 744-5952
Penn. (215) 922-5564
Calif. (213) 689-9324

CanPet '90

CANADA'S LARGEST AND BEST ATTENDED PET SUPPLY SHOW

CanPet '90 . . .

A WORLD-CLASS SHOW, IN A WORLD-CLASS VENUE HOSTED BY A WORLD-CLASS CITY

WEDNESDAY, SEPTEMBER 12
FROM 10:00 am–8 pm
THURSDAY, SEPTEMBER 13
FROM 10:00 am– 6 pm

New venue . . .

NOW ALL ON ONE FLOOR AT THE SPACIOUS INTERNATIONAL CENTRE, 6900 AIRPORT ROAD, TORONTO

Show Features:

FREE PARKING
FREE ADMISSION
TO BONA FIDE TRADE BUYERS
EDUCATIONAL SEMINARS SPONSORED BY THE CANADIAN PET INDUSTRY ASSOCIATION (CIPA)
EXPANDED EXHIBITOR SERVICES
1990 SPACE AVAILABLE AT 1989 PRICES
SPECIAL HOTEL RATES FOR ATTENDEES AT FIRST-CLASS ACCOMMODATIONS CLOSE TO THE SHOW SITE.

BE PART OF CANADA'S BEST SHOWCASE FOR PET SUPPLIES
. . . CANPET '90

FOR FURTHER INFORMATION PLEASE CONTACT;
FRED PEDDIE, SHOW MANAGER
OR, GREG WILLIAMS,
SENIOR PROJECT CO-ORDINATOR
THE CANADIAN PET SUPPLY SHOW
21 Lorraine Gardens, Islington, Ontario, Canada M9B 4Z5
Tel: (416) 231-9992 Fax: (416) 233-9725