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<th>ANIMAL GROUP AFFECTED</th>
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<td>Primates</td>
<td>Contact with food and bedding or housing contaminated by infected faeces</td>
<td>Mild or severe diarrhoea, fever, dehydration, collapse</td>
<td>Yes, in young animals</td>
<td>Fluids, electrolytes, antibiotics</td>
<td>In houses Good hygiene during food preparation and thorough cleaning of houses in zoos quarantine and regular screening, reduction of stress</td>
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**Susceptible animal groups**
Shigellosis is mainly found in captive primates in a zoo environment. However, in endemic areas, other mammalian species may also carry it. It has been found in freshly caught and newly imported animals, probably infected by human contact after capture. It seems to be more common in Old World apes and monkeys than New World species. It has been found in wild gorillas habituated to human visitors.

**Causative organism**
Shigellosis is caused by four species of Shigella organisms: *Sh. dysenteriae* (15 serotypes), *Sh. boydii, Sh. sonnei* and *Sh. flexneri* (several serotypes). The last species is most frequently found in primates. The organism colonises mostly the colon, sometimes the stomach, and occasionally extra-intestinal tissues. *Sh. sonnei* causes relatively mild clinical disease, *Sh. flexneri* and *Sh. boydii* are more virulent causing moderate to severe clinical disease and *Sh. dysenteriae* causes the most severe clinical signs.

**Zoonotic potential**
*Shigella* spp. can be transmitted to humans easily. *Sh. dysenteriae* causes dysentery in humans, but not in monkeys. *Sh. flexneri* 4b is regarded as almost exclusively a simian type. Affected people suffer from fever, diarrhoea, weakness and lethargy. The severity of the clinical signs depends on which *Shigella* sp. is involved, and on the age of the person (immune status) – adults are usually more resistant than children, except for immuno-deficient individuals.

**Distribution**
Animals: world-wide among captive primates.
Humans: human beings from mainly tropical and subtropical regions serve as natural reservoir.

**Transmission**
Faecal-oral route. Infected animals will excrete the organisms and contaminate the floor, walls of houses and the grounds of outside enclosures. Asymptomatic carriers may occur. Shedding in human carriers may persist for up to 17 months, whereas in recent infections excretion lasts for only days to a few weeks.
All bedding should be regarded as infected. Flies and other insects can be a major source of transmission. Keeper staff should be aware that they might carry infectious material on their shoes, clothing, and/or equipment (e.g. shovels, brooms, buckets etc.) used in primate houses. It can easily be transmitted from one primate house to another.

**Incubation period**
1-4 days.
Clinical symptoms
Clinical disease is often precipitated by stress (introduction of new animals in an existing group). Affected animals often show a day’s vague illness with loss of appetite prior to the development of diarrhoea (liquid or semisolid) and sometimes fever. Young animals are particularly affected, usually during first 2 years of life and may become chronic carriers. Severely affected animals have tenesmus, mucoid and sometimes blood-stained faeces, and become progressively weaker until they are semicomatous and lie prostrate in their cages. Diarrhoea may last several days or a few weeks in untreated animals, which may then become symptomless carriers. Dehydration and electrolyte loss is a serious consequence of illness. Morbidity is usually 20 %, but can reach 60% in some shipments. With therapy mortality can be held to about 5 %, but severe disease in overcrowded animals has caused deaths in more than 50% of newly arrived animals. Extraintestinal Shigella spp. infections of nonhuman primates appeared as polyarthropathies, gingivitis, gastritis, air-sacculitis or abortion.

Post mortem findings
Gross lesions:
Acute bacillary dysentery: edematous ileocecal valve, edematous mucopurulent or mucohemorrhagic colitis, congested and enlarged spleen
Chronic lesions: thickening and ulceration of colon and distention of lymphoid aggregates, sometimes strictures.
Microscopic lesions:
Acute lesions: destruction of superficial epithelium (may appear gangrenous), exudation of fibrin and neutrophils, crypt abscesses, mucosal and submucosal vessels contain fibrin thrombi and marginated neutrophils
Chronic: herniation of crypts into lymphoid patches with crypt ectasia and crypt abscesses.
Septicaemia is rare even with invasive strains. An ulcerative gingivitis and periodontitis has been described with Sh. flexneri in rhesus and bonnets with regression and hyperplasia and 50% of affected animals will shed Sh. flexneri in faeces. Gastric lesions consisted of mucosal haemorrhages or fibrinous-ulcerative inflammations particularly of the fundic and antral mucosa.

Material required for laboratory analysis
Fresh faeces or rectal swabs.

Relevant diagnostic laboratories
Diagnosis
Bacterial culture of fresh materials or of materials shipped in suitable transport media (Cary-Blair-medium, Teague-Clurman-medium). Shigella spp. are gram-negative, non-motile, non-spore forming slender bacilli that are relatively easy to grow from samples of fresh stool or rectal swabs. Single bacteriological surveys will not detect all carriers. Use a transport medium for swabs and inoculate on McConkey’s agar, Endo-agar, Salmonella-Shigella-agar, EMB-agar, or XLD-agar at 37°C for 24 hours.

Treatment
Affected animals should be given fluids and electrolytes and appropriate antibiotic treatment based on an antibiotic sensitivity test, since Shigella sp. can rapidly develop antibiotic resistance. When suspected, all in-contact animals should also be tested and possibly treated as well.

Prevention and control in zoos
There is no vaccine licensed for use in zoo primates, but in humans a streptomycin-dependent vaccine given orally 3 or 4 times has provided protection for 6 to 12 months. Vaccines provide partial protection for 3 to 6 months to non-immune individuals in highly endemic areas. However, the routine use of vaccines is not recommended.

Control in animal housing can be summarised as follows (lets separate this point into after disease occurs and prophylaxes to prevent infection of other animals. It might be easier to establish certain guidelines for general husbandry rules or cite the appropriate manuscript and only focus on specific measurements.):
1. Isolation and treatment of sick animals and unapparently infected permanent excretors of Shigella
2. Cleaning and sterilisation of cages, pens and houses
3. Prevention and reduction of stress (over-crowding)
4. Proper disposal of contaminated bedding and waste, and control of rats and flies etc.
5. Only keepers and other essential staff (with protective clothing, use of mouth mask) should enter infected premises. Non-essential visitors should be banned.
6. Regular bacteriological faecal testing in primate staff.

Control in zoos is best achieved by strict quarantine regulations for new arrivals and aggressive treatment or disposal of infected animals within the collection.

Suggested disinfectant for housing facilities
Notification
When *Shigella* organisms are isolated, the staff should be informed of the risks of infection, and appropriate health and safety measures put in place. In most countries, public health authorities have to be informed by the diagnostic laboratory about positive findings (especially for human cases).

Guarantees required under EU Legislation

Guarantees required by EAZA Zoos

Measures required under the Animal Disease Surveillance Plan

Measures required for introducing animals from non-approved sources

Measures to be taken in case of disease outbreak or positive laboratory findings

Conditions for restoring disease-free status after an outbreak

Relevant diagnostic laboratories

Contacts for further information

References