Hazard Identification

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HAZARD IDENTIFICATION

Hazard identification is a categorization step, identifying biological agents dichotomously as potential hazards or not. The risk assessment is concluded if hazard identification fails to identify potential hazards associated with the importation. The criteria employed for identifying hazards for imported animals and animal products are listed below:

The identification of hazards for the importation of animals and animal products must be in accordance with the Sanitary Phytosanitary Agreement of the World Trade Organization.

The hazard identification involves identifying the biological agents exotic to the importing country (including foreign strains, serovars, serotypes, species, or sub-species) or represent biological agents of diseases for which national control and eradication programs are in-place and which could potentially produce adverse consequences associated with the importation of a commodity.

The potential hazards identified would be those appropriate to the species being imported, or from which the commodity is derived, and which are present in the exporting country in that species or other susceptible species.

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A disease agent is only a hazard for that commodity if the agent can infect or contaminate the commodity, can survive any treatment and transportation, and potentially be exposed to a susceptible host (primary or secondary) resulting in adverse consequences.

The Office international des épizooties (OIE) list of diseases called List A and List B and the Food and Agriculture Organization (FAO) List C represent the principal lists of diseases (biological agents) for conducting hazard identification for the importation of animals and animal products.

The hazard list may include those vector-borne diseases for which there exists no known competent vector in the importing country. The potential adverse consequences would result from and be limited to disease in the imported animals themselves.

With respect to animal products, the disease agents must be able to survive any processing methods, the time-interval between harvesting/processing and importation, and then be exposed to a susceptible host. This combination of processing, mode of transmission, and exposure to target host or hosts greatly reduces the list of hazards associated with animal product importation.

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Exposure of a susceptible host to a hazard in an imported product would occur through the oral route. The deliberate feeding of product in swill or as uncooked scraps to swine, or as scraps either deliberately fed to, or foraged by, dogs, results in these two species being the major target hosts exposed to hazards. The use of imported feather, meat, bone and blood meal as a dietary supplement (as a mineral lick or as part of a compounded feed) or in fertilizer exposes additional target hosts.

The hazards identified for feather, meat, blood and bone meals reflect the likelihood of recontamination of these products with raw material following rendering at temperatures that may be more than sufficient to inactivate many animal pathogens.

The evaluation of the veterinary services, surveillance programmes and zoning and regionalisation systems may be important inputs for hazard identification with respect to the presence of a biological agent infecting an animal population in the exporting country.

Animals and animal products being imported into internationally recognized zones in the importing country which are free of specific diseases (biological agents) would necessitate that these disease agents be considered as hazards.

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Special considerations for imported zoo and wildlife species

Identification of hazards for zoo and wildlife species presents a challenge because of the lack of scientific information about specific diseases for many zoo and wildlife species. Evidence of antibodies on serology, in the absence of natural infection, may not be sufficient to call a pathogen a hazard. This is particularly relevant for carnivores and omnivores. The prey that these species consume may be infected with a variety of pathogens resulting in seroconversion (spill-over effect), yet may not be transmissible to other animals or humans.

Identification of an pathogenic agent as a hazard for the zoo or wildlife species under consideration requires:

E_1 evidence of natural infection and/or recovery of the pathogenic organism,
E_2 consideration of the pathogenesis and epidemiology of the disease, and
E_3 consideration of the phylogenetic relationship between known susceptible species and the species of zoo or wildlife under consideration

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Embryos
The International Embryo Transfer Society (IETS) has developed recommendations in its Manual (third edition 1998) in order to mitigate the disease risks associated with the transfer of embryos produced both in-vivo and in-vitro. The procedures detailed in Recommendations for the Sanitary Handling of Embryos in Chapter III of the IETS Manual are internationally recognised and followed, and hazards which have been shown to be reliably removed or inactivated by adherence to these procedures are not considered as animal health hazards. The IETS classifies pathogens into four categories according to the risk of transmission:

Category 1 risk of transmission negligible provided embryos are properly handled
Category 2 risk is negligible but status being verified by further work
Category 3 preliminary evidence is that the risk is negligible, but further work required
Category 4 preliminary work in progress

Diseases listed in Categories 1 and 2 are not considered as animal health hazards. The Research Subcommittee of the IETS Import/Export Committee reviews current research periodically and the categorisation may change as a result.

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Trypsin treatment
Washing of embryos with trypsin has been shown to be effective in removing or inactivating certain viruses which appear refractory to the standard washing procedures. These pathogens are retained as hazards as a reminder to specify trypsin treatment, which represents a modification of the standard protocol.

In-Vitro Fertilized Embryos
Differences in the zona pellucida invalidate the treatment of risk presented by IVF-derived embryos as equivalent to that presented by in-vivo fertilized embryos. (e.g., efficiency of trypsin treatment in eliminating pathogens from IVF embryos has not been demonstrated, therefore its use would be empirical). For this reason IVF embryos are treated as a separate category.

Micro-Manipulated Embryos
Provided micro-manipulated (e.g., sexed, cloned) embryos are in-vivo derived and treated using standard washing procedures prior to manipulation, they may be regarded as equivalent to non-manipulated embryos.

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Semen
Agents inactivated or removed from semen by internationally recognised collection, processing and storage methods (which detail dilution procedures (extension) and addition of antibiotics in order to mitigate the associated risk of transmission of disease) are not considered as animal health hazards.

Semen and embryos for import into special health status populations, such as artificial insemination centres, may result in additional hazards being identified. Responsibility for requesting health assurances mitigating these additional risks must rest with the management of the facility in question.

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Veterinary Biologics

The production of veterinary biological products involves processes which are subject to variability and source materials which, by their nature, provide good substrates for the growth of microbiological contaminants. A large number of different hazardous agents may potentially be associated with imported veterinary biologics, dependent upon the particular product under consideration.

Consideration of the country, region or zone of origin of each component of the veterinary biologic is of great importance, particularly regarding non-traditional disease agents such as prions which are unlikely to be inactivated or removed by the manufacturing process. The components which present the most risk are those substances of animal origin which are used to supplement media in which the agent undergoes amplification. Other components which may introduce a hazardous agent include substances of animal origin used in diluent, master seed strains (that is, live bacteria, viruses or parasites), specific-pathogen-free (SPF) eggs used for amplification of agents used in live vaccines, cell substrates upon which pathogens are grown, and water.

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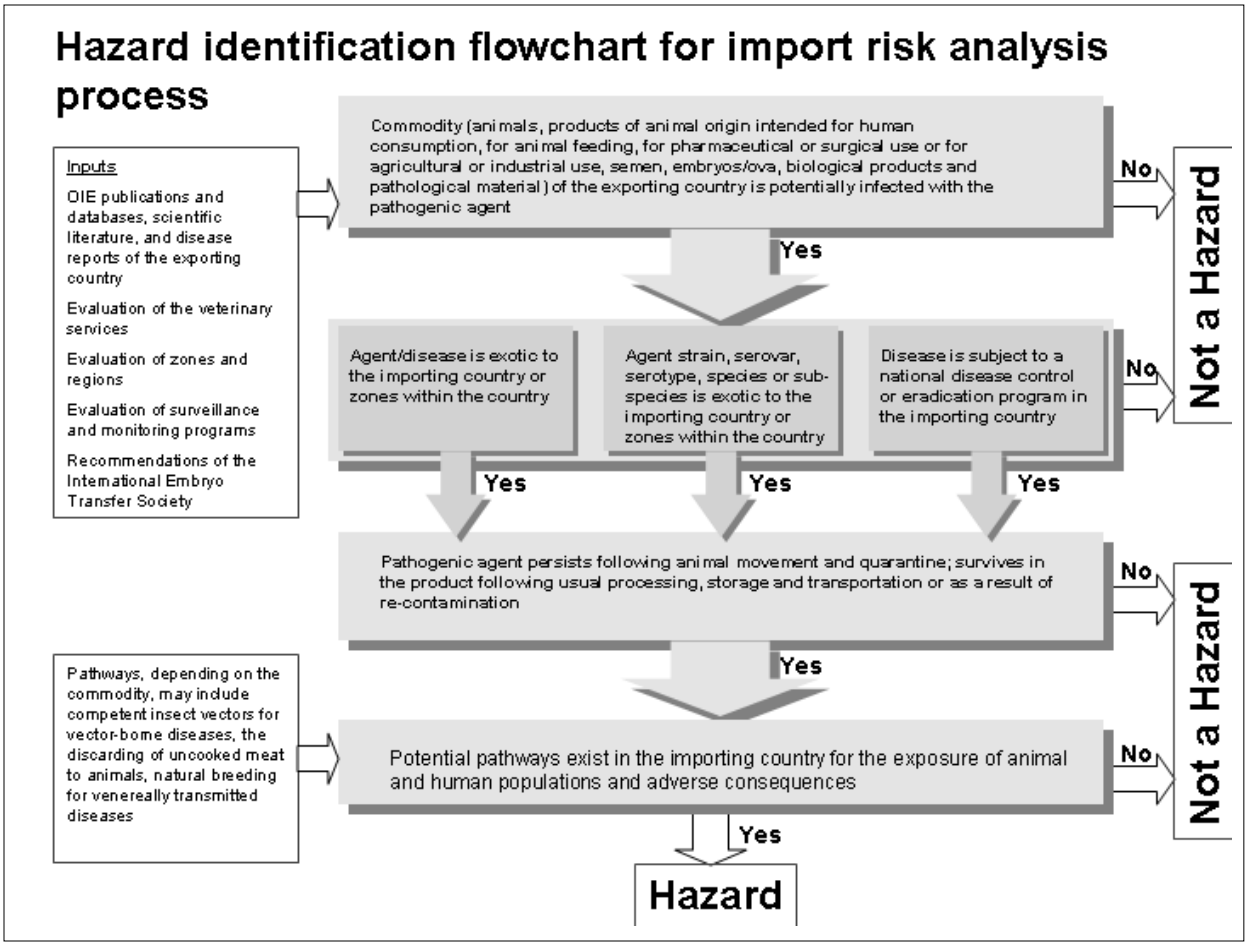
Veterinary Biologics

Good manufacturing practice (GMP) is particularly important in reducing the hazards associated with veterinary biologics by attempting to eliminate defective environmental conditions which could allow contamination of the biologic during the production process. This is most likely during the so-called "open stages", prior to sealing of the product into sterile vials or ampoules. These stages include formulation (where adjuvants, stabilisers and preservatives are added), filling, and freezing. Inadequate inactivation of any immunogen and cross-contamination with other products manufactured at the facility are also risks. The "closed stages" of production include the labelling and storage of the biologic - incorrect identification at this stage will cause inappropriate use of the product later.

In conclusion, the hazard identification for imported veterinary biologics evaluates:

- **vaccine microorganism or immunogen and its source**
- **country or zone of production facility**
- **other biological agents in vaccine facility and their source**
- **animal origin reagents and their source (e.g.. fetal bovine serum, bovine serum albumin, media containing peptones and protein hydrolysates of bovine origin)**
- **cell substrates (cell lines, primary cells, SPF eggs) and their source**
- **process (e.g., culture, concentration, harvest, separation, blending).**



Hazard identification for the importation of Paso Fimo horses (mares, stallions and geldings) from Colombia into Canada, May 2000.

HAZARD IDENTIFICATION

Hazard	Occurrence in Colombia	Occurrence and Control Measures in Canada	OIE List
VIRUSES			
Vesicular stomatitis virus	+	(1949) *Qf	A020
Rabies virus	+	+ * Qi	B058
Equine infectious anemia virus	+	+ * Qf Sp Qi Te	B205
Venezuelan equine encephalomyeltis virus	+	0000 Qf	B216
RICKETTSIAE			
BACTERIA			
Bacillus anthracis (anthrax)	(1998)	+ * Qi V	B051
Leptospira spp. (foreign serovars)	+	+	B056
Brucella abortus	+	+ * Qf Qi Su Te Vp	B103
Salmonella abortus equi	(1989)	0000	C754
PROTOZOA			
Babesia caballi, Theileria equi(piroplasmosis)	(1996)	*(1987) * Qf*	B207
Trypanosoma evansi (surra)	1990	0000 Qf	B215
PARASITES			
Chrysomia bezziana (screwworm)	-	0000	B060
Psoroptes equi	-	0000 * Qf	B213
Parafilaria multipapillosa (filariasis)	(1993)	0000	C622
Integumentary athropods (foreign)			-

Disease Occurrence:
0000 disease never reported
(month/year) date of last reported occurrence in previous years
+ reported present or known to be present
- disease not reported (date of last outbreak not known)

Disease Control Measures:
*** - notifiable disease**
Cn - control of arthropods
Cr - control of wildlife reservoirs
M - monitoring
Qf - precautions at the border
Qi - movement control inside the country
S - stamping out
Sp - modified stamping out
Su - surveillance
Te - screening
V - vaccination
Vp - vaccination prohibited
Z - zoning

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Hazard identification for the importation of swine embryos into Canada from Japan, June 2000.

HAZARD IDENTIFICATION

Hazard	Occurrence in Japan	Occurrence and Control Measures in Canada	OIE List
VIRUSES			
Foot and mouth disease virus	(3/2000)	(1952) * Qf	A010
Classical swine fever virus	(12/1992)	(1963) * Qf	A130
Pseudorabies virus (Aujeszky's disease virus)	+	0000 * Qf Qi S	B052
RICKETTSIAE			
BACTERIA			
Brucella abortus (bovine brucellosis)	(1992)	+ * Qf Qi S Te Su Vp	B103
Mycobacterium bovis (bovine tuberculosis)	+	+ * Qf Qi S Te Su	B105
PROTOZOA			

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Hazard identification for the importation of deboned beef into Canada from Argentina, January, 1996.

HAZARD IDENTIFICATION

Hazard	Occurrence in Argentina	Occurrence and Control Measures in Canada	OIE List
VIRUSES			
Foot and mouth disease virus	(04/1993)	(1952) * Qf	A010
RICKETTSIAE			
BACTERIA			
PROTOZOA			
PARASITES			

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Radostits, O.M., Blood, D. C., Gay C. C. Veterinary Medicine. A Textbook of the Diseases of Cattle, Sheep, Pigs, Goats and Horses. 8th edition, 1994. 1763 pp.

HAZARD IDENTIFICATION

Newcastle disease (ND) is an infectious, highly contagious and pathogenic viral disease, which affects chickens, turkeys and many other domestic and wild bird species. Occasionally humans are affected. ND virus (NDV) is an enveloped RNA virus, a member of the Paramyxoviridae family, Paramyxovirinae subfamily and Rubulavirus genus (Alexander 1997).

pH Stability

The pH range of stability is broad and NDV tolerates pH 2 to 10 (Beard and Hanson1984). The infectivity of the virus is not appreciably modified between pH 4 and 11. It loses some of its infectivity at pH 3 and almost all at pH 1 and pH 13 (Commission des Communautes Européennes 1975).

Thermal stability

Thermal stability depends on the strain but all activity is destroyed at 100 degrees C for 1 min. At 56 degrees C destruction of infectivity, haemagglutinating activity and immunogenicity occur within 5 min to 6 h. At 37 degrees C, hours and days may be required and at lower temperatures (20 and 8 degrees C) the virus can be stable for months and years (Beard and Hanson 1984).

Hazard identification for the importation of ostrich meat into Canada from South Africa, February 1999

In trials in which the survival of 4 NDV virus strains at differing times and temperatures was assessed it was found that: -
At 70 degrees C virus was inactivated within 40 to 50 sec.
At 60 degrees C virus was inactivated within 6 to 7 min.
At 37.5 degrees C virus was inactivated within 8 to 11 days.
At 22 degrees C (ambient temp) virus was inactivated within 25 to 42 days.
The authors concluded that all strains, whether virulent or avirulent, showed no difference in viability (Foster and Thompson 1957).

Ultraviolet ray sensitivity

Ultraviolet rays destroy NDV similarly to other myxoviruses (Beard and Hanson 1984). The virus is inactivated in 35-45 minutes by a wavelength of 2537D (254 nm) and in 0.8-1.08 seconds by a wavelength of 1600-1800 D (160-180 nm) (Brandly and others 1946). The sun emits a wide variety of electromagnetic radiation, including infrared, visible, ultraviolet A (UVA; 320 to 400 nm), ultraviolet B (UVB; 290 to 320 nm), and ultraviolet C (UVC; 10 to 290 nm). The only UVR wavelengths that reach the Earth's surface are UVA and UVB. UVA is the predominant ultraviolet light reaching the Earth's surface (tenfold to one hundredfold more than UVB) (National Institutes of Health 1989)

Environmental stability

Stability of the virus in the environment depends on the medium in which it is present; carcasses, faeces, mucus, decaying materials, proteinaceous matter. Warm temperatures and solar radiation facilitate destruction of NDV (Alexander 1980). Infectious virus may survive for months at room temperature in eggs laid by infected hens, and for over a year at 4 degrees C. Similar survival times have been observed for virus on feathers, and virus may remain infectious for long periods in contaminated premises (Fenner et al. 1987). In an examination of the ability of NDV to survive in fermented edible waste material, it was found the NDV survived the entire test period at temperatures of 5 to 30 degrees C (Wooley et al 1981; Beard and Hanson 1984). In a further study of the antimicrobial effects of Lactobacillus fermentation survival of NDV in infected chicken carcasses was examined in waste material. In two trials NDV survived 4 days at 20 degrees C, 2 days at 30 degrees C and 1 day at 40 degrees C (Shotts et al 1984).

Hazard identification for the importation of ostrich meat into Canada from South Africa, February 1999

Host Range

- natural or experimental infection with NDV has been demonstrated in at least 236 species from 27 of the 50 orders of birds (Alexander 1997)
- chickens, turkeys, pigeons, guinea fowl, peacock, pheasants, quail, partridges (Kouwenhoven 1993)
- geese and ducks are usually regarded as resistant even to NDV strains most virulent for chickens (Alexander 1997)
- ostriches can become infected (Samberg and others 1988; Huchzermeyer and Gerdes 1993)
- wild birds represent a potentially important but unknown reservoir
- although people may become infected with velogenic viscerotropic Newcastle disease (VVND) virus, the resulting disease is usually limited to conjunctivitis. Recovery is usually rapid and the virus no longer present in eye fluids after 4 to 7 days; infections have occurred mostly in laboratory workers and vaccinating crews; no instance of transmission to humans through handling or consumption of poultry products is known (Alexander 1991).
- 1971-1973 California outbreak involved 391 flocks (86% chickens, 6% exotic birds, 3% pigeons, 2% game birds, 2% turkeys, 1% ducks and geese (Burridge 1975)
- an added complication in the epidemiology of VVND was experienced in outbreaks in Great Britain during 1984 when virulence of the virus from pigeons increased within respect to chickens, only after passage through chickens (Alexander 1985)

Global Distribution

- ND has been reported worldwide: Europe, Asia, Americas, Africa, Japan, and Australia
- in most countries with developed poultry industries, lentogenic and some mesogenic forms are
common, the velogenic forms (viscerotropic (VVND) or neurotropic (NVND)) are less common
- Alexander (1997) considered three panzootics of ND: 1) slow, worldwide spread from Southeast Asia to Europe in poultry from 1926 to the 1960s, 2) rapid spread from the Middle East to worldwide in the late 1960s to 1973, believed to have originated from imported caged psittacine birds which continued to be an important factor in the spread of the disease, and 3) rapid spread from the Middle East in the late 1970s to the early 1980s, originating from pigeons and doves including the spread to chickens in Great Britain in 1984, through feed that had been contaminated by infected pigeons.
- examples of specific outbreaks of VND include:
- poultry in California 1971-1973, of which the source of infection was considered to have been illegally imported exotic birds (Burridge and others 1975)
- poultry in England 1970-1972 and 1984 (Alexander and others 1984)
- imported cockatoos and lovebirds appear to have been a source of VVND in Japan in 1980 (Hirai 1981)
- exotic pet birds in five American States April-July 1991; eradicated without spread to domestic poultry; source: Amazon parrots suspected of being illegally imported into Texas (Brunning-Fann and others 1992)

Hazard identification for the importation of ostrich meat into Canada from South Africa, February 1999

- EU countries 1986-1990, 85 outbreaks, 75 in Italy including 45 in 1988, and trend of increasing EU numbers from 18 in 1991, to 83 in 1992, to 134 in 1993; about 40% of these EU outbreaks have been in hobby birds (Alexander 1995)
- recently in Canada outbreaks of ND in wild cormorants, pelicans, gulls and terns have occurred in 1990 and 1992, with isolation of velogenic virus and neurologic clinical signs in wild birds, but no evidence of transmission to commercial poultry (Wobeser and others 1993, OIE 1990; 1992)
- VND was confirmed in a range flock of 26,000 turkeys in North Dakota in 1992, demonstrating neurological symptoms and located approximately three miles from where a 'die-off' of cormorants had occurred (Grow 1992, Meteyer and others 1997).

Modes of Transmission

- transmission occurs by direct contact via ingestion of infective material or inhalation of excreted droplet particles; the success of the inhalation route of transmission will depend on many environmental factors such as temperature, humidity, and stocking density (Alexander 1995)
- vertical transmission is controversial; its true significance not clear; transovarial transmission may be important especially with lentogenic strains, and virus infected chicks may hatch from virus-containing eggs; cracked or broken eggs, or eggs contaminated with faeces can be a source of virus for newly hatched chicks (Alexander 1997).

Hazard Identification *CFIA/ACIA*

Spread

- inapparently infected carriers are the most likely source for introduction of VVND into ND free countries, including numerous species of exotic pet, game and exposition birds, racing pigeons, waterfowl and domestic poultry (Alexander 1995)
- many species of caged birds harbour VVND without showing clinical signs, so the smuggling of captive birds poses a hazard (Alexander 1988)
- wild cormorants may have been the source of infection for an outbreak of velogenic ND among range turkeys in North Dakota 1992 (Grow 1992), however, NDV isolates from migratory birds are usually of low virulence (Alexander 1995)
- the 1971-73 California outbreak experienced extensive spread between flocks by movement of live birds and mechanical transport of virus by vaccination and service crews on clothes and equipment; there was no evidence of significant wind-borne spread in that outbreak (Burridge and others 1975)
- 19 of 23 outbreaks in Great Britain between February and July 1984 occurred directly or indirectly as a result of spread from diseased pigeons infecting feed stores at port docks (Alexander 1985); the preparation of food for layer and broiler-breeder flocks involved no process which would adversely affect virus infectivity (Alexander 1985)
- outbreaks in Great Britain in the early 1970's indicated windborne spread of up to 8 km.; but other outbreaks (e.g., California 1971, England 1984) appeared not to involve windborne spread (Alexander 1991)
- human beings can become infected with NDV resulting in a conjunctivitis but man's involvement in the spread of ND is most likely by the transfer of infective feces on the hair, clothing, footwear, crates, feed sacks, egg trays or vehicles (Alexander 1995)
- mechanical transfer of infective feces by insects, rodents and scavenging animals may occur (Alexander 1995)

Hazard identification for the importation of ostrich meat into Canada from South Africa, February 1999

Incubation Period

- the incubation period of ND after natural exposure has been reported to vary from 2 to 15 days (average of 5-6 days) (Alexander 1997).
- incubation in natural infections is 4-6 days (Fenner 1993)
- neurotropic velogenic Newcastle disease (NVND) virus isolated from racing pigeons in Sweden caused high mortality and a incubation period of 5-11 days in chickens (Engstrom 1985)
- the mean death time for inoculated pigeons was 9.5 days (range 4-25) and virus was shed for up to 20 days (Pearson 1987)

Clinical Disease

Pathogenesis