AN AVOIDABLE EPIDEMIC OF OPMV IN COLLECTIONS OF AUSTRALIAN SNAKES AND THE WIDER IMPLICATIONS OF THE DISEASE IN AUSTRALIA AND ELSEWHERE.

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Note dated 23 July 2003: The following is a draft paper only completed some weeks back. It does not take into consideration the Electron Micrograph Results of end July 2003. Statements by John Weigel in a post dated 22 July 2003 at http://www.acay.com.au/~dunnwell/reapz/reptile.htm provides no new information of relevance and does not in any way affect the conclusions below - rather they confirm the facts as known.

ABSTRACT

This paper reports on a case of ophidian paramyxovirus (OPMV) in a collection snakes in Victoria, Australia and a wider epidemic in several other Australian collections. Further reported is the history leading to the first diagnosis, including the means of infection, location of the original source of the infection and by investigation the source of that, further OPMV infections from these sources, a history of other known cases in Australia and the ramifications of this disease in terms of captive-held collections and potential escape into the wild. This paper notes the adverse consequences of failure to report infection to persons who received infected snakes.

Unlike most other reports on OPMV that report in detail on the histopathology and microscopic properties of the virus, this paper details the case from the perspective of a reptile keeper in terms of observation of symptoms and diagnosis and in view of alerting other keepers of these indicators of infection.

This paper refutes claims that the primary means of transmission is airborne. It is not. Fluid-based transmission is demonstrated. For the first time ever, this paper explains previously noted differences between the progression of OPMV in situ and in the laboratory. There are also numerous pointers as to which OPMV affected snakes will show symptoms and die and those which may not or instead by asymptomatic carriers.

Evidence is also presented that indicates the following: OPMV is particularly deadly to small snakes, effectively stops growth in affected small snakes, is often carried by large snakes without apparent symptom, is probably a recent import into Australia and other new findings.

OPMV infections can also be effectively tracked though collections, even in the absence of detected symptoms, based on known cases within one or more collections, transaction histories and an appraisal of management practices within collections.

OPMV AS KNOWN TO DATE

Most respiratory infections in reptiles have been traditionally thought of as bacterial in origin. Such infections have been regarded as opportunistic explosions of (often natural) bacterial flora (including the likes of *Pseudomonas*, *Aeromonas*, etc) as a result of stress in captive reptiles, including poor husbandry, excessive handling and in particular cage temperature being too cool.

An example of this prevailing thinking was seen in Banks (1980) who at the time wrote:

> '*Respiratory infection is usually the result of a sudden decrease in temperature, but it may also be brought on by overcrowding or stress conditions when the animal's resistance is lowered*.'

Treatment of these infections, as indicated by texts such as Banks (1980), has generally been by removing the husbandry and related problems alleged to have caused the infection, such as cage temperature, other specimens, substrate, etc, as well as anti-biotic treatment to attack the bacterial infection and if possible the raising of the cage's temperature so as to enable the reptile's own immune system to work to it's full potential.

In 1976 a respiratory epizootic spread through a collection of fer-de-lance (*Bothrops atrox*) at a snake farm in Switzerland (Foelsch and Leloup, 1976). Although *Pseudomonas* and *Aeromonas* were isolated from the respiratory tracts of dead snakes, a virus (FDLV) with ultrastructural properties similar to the myxoviruses (ortho-, para- and meta-) was identified (Clark and Lunger, 1981). That the FDLV is a paramyxovirus was indicated by demonstrating it possessed a single-stranded RNA genome and had a sedimentation value of 50S (Jacobson, 2000).

In the past decade and in light of this new information, OPMV associated die-offs have been identified (either by virus isolation and/or characteristic histopathologic changes) in a variety of private and zoological collections in the United States (Jacobson, 2000).

Paramyxovirus has also been identified in lizards (Nichols, et. al. 1998).

Histopathological changes in reptiles suffering Paramyxovirus are now well documented in the scientific literature as is the progress of OPMV infections in snakes in the laboratory situation.

Paramyxovirus as identified in reptiles is believed to be from an undescribed genus.

In terms of the husbandry and conservation aspects of OPMV in lizards, the same applies as for snakes. However for the purposes of this paper, OPMV in snakes only is considered.

Based on the reports of Jacobson (2000) and others, it appears that there are multiple forms of OPMV and some which appear to target particular species within a collection and leave other snakes apparently untouched and/or asymptomatic, meaning that the snakes carry the virus but don't show clinical signs of infection.

Contrary to assertions by John Weigel (personal phone communication 3 July 2003), the mere fact a snake does not show signs of OPMV infection does not mean it isn't a carrier. Experiences in terms of the epidemic detailed in this paper adequately prove this point.

Jacobson (2000) wrote:

> *'In one collection having at least 12 crotalid species, the major species affected was the Mexican west coast rattlesnake (*Crotalus basiliscus*). In another die-off, a long term collection of 30 to 40 north Pacific rattlesnakes,* (Crotalus viridis oreganus)*, died over a 1 to 2 month period. In 1987 a major die-off of Mojave rattlesnakes,* (Crotalus scutulatus)*, in a research collection was attributed to PMV infection. In 1988 PMV was isolated from a black mamba (*Dendroaspis polylepis*) in a serpentarium experiencing a die-off of viperids, elapids, boids, and colubrids. In 1988, PMV was also isolated from clinically ill corn snakes (*Elaphe guttata*), beauty snakes (E. taeniurus), and Moellendorff's ratsnakes (*E. moellendorffi*). In the Federal Republic of Germany, a myxovirus- like agent was recovered from a red-tailed rat snake (*Elaphe oxycephala*) (Ahne et al., 1987).*'

The symptoms of OPMV in snakes varies, but in most diagnosed cases the most obvious target appears to be the respiratory tract.

This variation is between individual snakes and species. However there are some distinct trends between species known to be infected with the same virus (see later).

In captive snakes OPMV is often seen manifesting as a respiratory infection with the lung cavity swelling and fluid being exuded through the mouth.

It usually appears in an apparently spontaneous way and in spite of no apparent change to caging husbandry and the like. The only change being another (infected) reptile being moved into the facility, although this factor is often overlooked by keepers.

(In the absence of indicators of other illness or known husbandry defect, these symptoms and factors should be taken as indicative of a likely OPMV infection).

Other symptoms of OPMV infection are known to include excessive urination (and drinking), often seen manifesting as a rapid emaciation of the snake and apparent loss of condition at abnormal speed, blood from the mouth and neurological signs. The latter manifest as a form of 'stargazing' or unnatural postures as seen in IBD in affected pythons and boas.

Based on published reports and my own experience (see below), OPMV usually, but not always acts quickly in affected reptiles and appears to go through four stages once it appears in a live reptile. Death usually results from 5-12 days after symptoms appear.
These stages are as follows:

- **Stage 1** - A loss of muscle tone and the reptile stretching out in a 'linear manner' with the head slightly raised. This stage may also manifest in "star-gazer" type behaviour, loss of vitality, unnatural poses and other more subtle cues.
- **Stage 2** - Lasting one to two days - The reptile displays unusual restlessness and holding the mouth open. It will apparently wander around the cage aimlessly. Tongues are kept in the mouth and pupils dilated.
- **Stage 3** - Seen from several hours to a day before death. This involves the mouth being kept open, sometimes only slightly and the snake will expel a pus-like material from the glottis.
- **Stage 4** - Seen from several minutes to one hour preceding death. The mouth is kept fully opened, the pupils are dilated, and reptile will remain excessively active, including exhibiting bouts of convulsive behavior.

Other than treatment of secondary bacterial infections with relevant antibiotics, such as Baytril and other general pallative care, published literature says that there is effectively nothing that can be done to help affected snakes (see later).
At the terminal phase of OPMV a snake will appear to be quite emaciated, even if it had appeared very healthy just days or a few weeks earlier. Put simply, the decline is rapid.
A hemagglutination-inhibition (HI) assay has been developed to measure antibody produced against OPMV (Jacobson et al., 1981), however in experiments, some snakes produced no antibodies some weeks after infection and others actually died before any antibodies appeared. Hence a negative HI assay cannot be taken to exclude the possibility an OPMV infection. It appears that in some snakes OPMV antibodies form about 8 weeks after infection. This may in part have explained the delayed cure of respiratory infection in an infected adult female *A. cummingi* and a case involving an infected Scrub Python (*Australiasis amethistina*) (see below).
The 8 week period is also important in terms of the commonly observed situation of affected snakes often regaining health after this period of showing OPMV-related symptoms.
The virus can therefore only be detected with certainty via electron microscopy (EM).
Diagnosis of the virus in situ is generally 'presumptive' and based on the history of the reptile, and after death by gross and histological examination of tissues including of the lungs, respiratory tract and associated organs, brain and kidney.
One of the aims of this paper is to aid in preliminary and presumptive diagnosis in situ to enable keepers to take rapid remedial action and reduce potential losses.
The information provided below also shows how OPMV infected snakes can be reliably predicted, based on known cases, movements of reptiles within and outside the collection as well as via an appraisal of husbandry methods.
Jacobson et. al. (1997) tested a vaccine for OPMV in western diamondback rattlesnakes (*Crotalus atrox*), but the results were disappointing, including death of vaccinated snakes and so an effective cure or preventative is not yet available.

KNOWN OPMV CASES IN AUSTRALIA

Posts circulated on the internet under the header "Out-of-Session Item No: 01/2003 Consultative Committee on Emergency Animal Diseases" (Christie, 2003) in January 2003 indicated presumptive diagnosis of OPMV in Australian collections of snakes.
This was an apparently leaked memo and sent out without the approval of the Australian Reptile Park owner John Weigel.
It listed infections, including presumptive diagnosis of snakes at the Australian Reptile Park (ARP), Somersby, NSW, based on a mass die off of Death Adders (*Acanthophis* sp.) starting February/March 2002, then abating somewhat and then re-appearing in the spring months to November. Whilst starting with Death Adders, the infection later spread to other snakes including Tiger Snakes (*Notechis*), King Brown Snakes (*Cannia*) and a Woma (*Aspidites*).
The report also stated that cases had been presumptively identified in private collections in Queensland. My own inquiries in June 2003 showed that these latter cases were diagnosed by Clayton Knight of the Deception Bay Veterinary Clinic.
The circular claims the disease was thought to be exotic to Australia and claims the first known cases of it in Australia.
In all cases the diagnosis had been by clinical, gross and histological examinations.
Christie, 2003 implied that arrangements were being made to send samples to Elliott Jacobson to confirm the diagnosis of OPMV, but as of 24 June 2003 (Jacobson 2003b), this had not occurred.

THE EVENTS BEFORE THE HOSER DIE OFF

In early 2002, three new born and two one year old (est.) Death Adders (*Acanthophis antarcticus*) were obtained and housed in a room of a bungalow. All were raised to adult size (55 cm or larger) within 12 months and excluding a severe mite infestation in mid year that was cleared up without casualty using pest strips (Dichlorvos based ones), the raising of the snakes was generally uneventful.
(For the record the mite infestation was completely my own fault and I was lucky that no snakes were lost as one of the Death Adders did decline sharply in condition as a result and took some time to recover).
All snakes were housed separately in plastic tubs and water-bowls in the cages were of a disposable nature and hence there was never possibility of transfer of waterborne vectors at any stage of cage cleaning.
All caging in the room is effectively identical in that each container has a warm end heated in the mid 30's (°C), heated via a thermofilm heat mat placed underneath, with the cooler end of the cage at room temperature (ranging under 20°C most of the time and never over 30°C, due to a thermostat controlled air conditioner in the same room).
Between these ends is a heat gradient that is steepest at the area equivalent to the edge of the thermofilm underneath.
This is usually where the snakes tend to rest.
Each cage has minimal furnishings and only enough to provide the snake's essential needs in terms of water, cover, and the like. Substrate is hardened sand forming a rock-like surface, which in turn has loose leaves on top. Typically the snakes thermoregulate by siting themselves at the appropriate point of the heat gradient.
Around Christmas 2002, a Queensland Carpet Python (adult) and a NSW Diamond Snake (Juvenile) (*Morelia* spp.) were moved into the room and kept in identical conditions. Unlike the Death Adders, which had no cover other than leaves, they each had an upturned pot to shelter under, which they used.
Both appeared outwardly healthy at the time and remained so. Their captive history was unremarkable.
On 15 February 2003, an adult male and adult female Top-end Death Adder (*Acanthophis cummingi*) were obtained from keeper Stuart Bigmore of Lara, Victoria and housed in the same room and under identical conditions. Both had been originally obtained from the wild, although they had been captive for some time.
Bigmore had held the male for 18 months and the female since at least 1997.
They both presented with numerous health problems, including parasitic (nematode) in the male as well as protozoan (green runny feces from amoebic dysentery), bacterial infections, including severe respiratory infections (fluids from mouth) and open lesions on the body (both snakes). The two snakes had previously been housed at opposite ends of the same room at the previous facility and the similarity of infections between the snakes, in spite of the caging arrangements went unnoticed at the time.
As above, these snakes were also housed separately and over coming weeks were given a cocktail of anti-biotic (Neosporin), anti-helminthic (Panacur) and anti-protozoan (Flagyl), usually administered in force-fed food items such as rodents.
At this stage, neither ate voluntarily. After initially appearing to recover slightly, the more emaciated male declined and died on 10 March 2003. The cause of death was believed to be linked to the systemic infection as both the dysentery and helminth problems had been removed and the snake had passed several 'good' feces based on rodent and other food. At the time of death the snake also had the apparently intractable respiratory infection.
After deciding not to autopsy the snake, the intact frozen corpse was lodged with the National Museum of Victoria on 14 March 2003 (NMV D71474).
The female lived. After six force-feedings, the surviving female ate voluntarily for the first time on 19 April 2003. She appeared to make a full recovery in terms of health and condition thereafter.
Notable points here are that the two above snakes were housed in

the same bank of shelves as the other snakes, although until deemed healthy, they were handled with separate hooks and feeding implements, the most notable being the forceps used to offer food to the snake.
This quarantining ended around the end of March 2003 when it was decided that the adult female *A. cummingi* was in perfect health save for her reluctance to feed.
A persistent respiratory infection was noted, but overlooked as being a 'hang-over' from her previous poor health and not thought likely to be transferable to the other snakes. The basis of this decision (in hindsight erroneous) was that the respiratory infection was thought to be an opportunistic bacterial infection derived from the natural flora of the snake's respiratory tract and therefore not contagious.
On 25 Feb 2003, four newborn (born 22 Feb) Top-end Death Adders (*Acanthophis cummingi*), acquired from Ballarat-based keeper Roy Pails were moved into the room and housed as for the other snakes. All were fed almost immediately and more-or-less until death (see below) grew rapidly and without incident. One of the males, was however slightly smaller than the other three and was generally more reluctant to eat in terms of 'assist feeding'. It was therefore generally force-fed (food literally pushed past the head and neck using forceps), rather than 'assist fed' whereby food is put into the mouth and the snake then finishes the feeding process.
The same in terms of general husbandry applied in terms of two newborn Death Adders (*A. antarcticus*) (born 21 Feb 2003), received from Sydney-based keeper Alex Stasweski that were moved into the room on 21 March 2003.
On 17 May 2003, 14 Red-bellied Black Snakes (*Pseudechis porphyriacus*) were placed in plastic cage boxes immediately adjacent to the other snakes in the same room. These snakes were literally in transit as part of a legal shipment from NSW-based keeper Rob Gleeson to keepers Fred Rossignolli and Scott Eipper in Victoria. The snakes remained in this position until taken away on 20 May 2003.
At the time the physical health of the *P. porphyriacus* ranged from good to emaciated and all had a severe mite infestation. The mites were killed off using pest-strips (dichlorvos-based) before the snakes were stored in the room and there was no evidence of transfer of live mites to the other snakes in the room.
On 19 May 2003, two newborn Barkly Death Adders (*Acanthophis hawkei*) (born 4 May 2003) and acquired from Brian Barnett of Ardeer, Victoria were placed in the same room in similar housing to that described above.
Both ate the next day, (the male by itself, the female by assist feeding) and like all the other young Death Adders, presented as being in perfect health.

THE HOSER DIE OFF

As the history of the die-offs is related below, the other various possible causes of death are given (as mooted at the time). This is so that it becomes evident to the reader how the diagnosis of OPMV was finally reached and why other possible causes of death were discounted.
On 25 May 2003, the female *A. hawkei* was found outstretched and dead in it's cage.
The snake appeared outwardly healthy and the death appeared to be literally without explanation.
The method of caging was questioned but discounted as the cause as the other snake in the identical adjacent cage remained healthy as had all other Death Adders raised in identical circumstance.
Attention was then drawn to the food given to the snake, This had been two fish.
Two logical theories were either the food being too large for the snake to digest or perhaps spines from the fish piercing the digestive wall and death resulting.
Most herpetologists told of this death, did without asking further questions advance these theories.
Both theories were immediately discounted myself.
Noting that by that stage over 100 fish had been fed to young Death Adders in the previous three months and without incident the anti-fish theory seemed to lack weight.
More importantly, the size and timing of the food also effectively ruled out the fish.
The fish that had been fed had been very small (as befitted the caution of a 'first feed'). The size and easy digestibility of the fish used had meant that they had passed out of the stomach in about 30 hours (based on this and other cases).
At the time the snake died it's stomach had been empty for some days.
The final conclusion at the time was that the death was via no known cause and that all other snakes should be monitored more closely.
On 20 April 2003 one of the young male *A. cummingi* (AC-4), the smaller one mentioned already, was found in it's cage having shed in a piecemeal manner. Noting that I'd been absent for three weeks, the snake was thought to have shed a week or so earlier.
By the stage the snake was found it had been moving around the cage for several days trying to remove the now severely encrusted skin. As a result it appeared exceptionally thin and emaciated.
The snake was soaked in luke warm water. This was initially for an hour and then several more, before it was possible to manually remove (using my hands) the remaining skin.
The cause of this skin retention wasn't known, but at the time was blamed on a combination of the substrate (hardened sand) and diet, (e.g. the previous 6 feeds totaling 14 *Gambusia* fish). The oily nature of the fish was blamed for this anomaly.
Following three more (very small) non-fish feeds that snake was returned to a dominantly fish diet until it became of a size large enough to feed on small (pink) rats.
The snake did not die and apparently made a full recovery.
In terms of diagnosing respiratory ailment in the young Death Adders, there was a factor that effectively masked it.
When feeding wet and oily fish to small snakes (including young Death Adders), fluids commonly run out of the nostrils.
This may encrust and as a result the snake may have trouble breathing through partially blocked nostrils. In other words this may mimic a respiratory infection.
As this was the situation with most of the smaller Death Adders, it's likely that several may have had respiratory complaints and gone unnoticed.
Notwithstanding this, on 20 May 2003, the three other young *A. cummingi* were switched over to a diet of exclusively rodents. The fourth was likewise switched to rodents exclusively on 27 May 2003.
On 14 June 2003, it was noticed for the first time that the other young *A. hawkei*, a female *A. cummingi* and one of the young *A. antarcticus* (AAA-106) had respiratory infections that were clearly not a result of the fish being used as food.
The same conclusion was also made in relation to the other young *A. antarcticus* and another young male *A. cummingi* (AC-3), but both had since apparently recovered.
The basis of this 'new' discovery was a rapidly worsening condition in the *A. hawkei*, which as a result of the death of the other *A. hawkei* had not been fed fish (see above). In fact it's respiratory condition was by far the worst of the three, well and truly ruling out fish as causative of respiratory complaint.
The other snakes displayed a slightly open mouth with some crusty exudate around the lips. By contrast, the *A. hawkei* had this as well as still wet fluid oozing from the mouth and a puffed up neck.
Furthermore, as indicated above, the period that the *A. cummingi* had been feeding on rodents (and not fish) indicated that the problem was respiratory and not fish related as first thought. The lack of fish in this snake's recent diet showed emphatically that the health problem was not related to the fish as mooted previously.
Assuming that it was a respiratory infection being dealt with, the caging of the snakes came under renewed scrutiny, but was again cleared as being more-or-less optimal.
Notwithstanding this, it was noticed that two of the snakes, the *A. hawkei* and the *A. cummingi* were restless and moving around their cages.
In the room the snakes were in, the temperature (in June 2003) fluctuated around the 11-18 °C mark. Noting that such temperatures are believed suboptimal for snakes experiencing respiratory infections those three snakes were relocated.
Caging remained the same, as in heat mat at one end of their cage, giving a region in the cage in the 30's (°C). However the room these snakes were in (but not the others) was set at 23°C by use of heater and thermostat, thereby preventing the snakes from ever being cooler, even if they rested in the coolest part of the cage.
All water bowls had Baytril added to combat the respiratory infection (widened to include all snakes a week later). The snakes weren't given medicine directly as they tend to drink frequently anyway and the drug is known to be rapidly absorbed into the system once

ingested.
Furthermore, while Baytril is a drug of choice against respiratory complaints in snakes, injection sites for the drug are known to become necrotic.
The other outwardly unaffected Death Adders were left untouched on the basis that they retained the good sense to straddle the heat mat and keep their body temperature at the preferred range of about 28-37 °C.
A continued evaluation of the husbandry regime of the snakes led to my concluding that if the respiratory infection was caused by a bacteria not normally present in the snakes then my use of the same forceps to feed and force-feed the snakes may contribute to it's spreading. Hence on 14 June 2003 my commencement of a policy of using separate feeding implements for outwardly infected and outwardly uninfected snakes.
The following morning the young *A. cummingi* was found dead in it's cage. The snake was in a loose circle and upturned, which incidentally was the same position that the adult male *A. cummingi* had been found in some months earlier.
The cause of death was presumed to be from one of two causes.
One was suffocation via the respiratory infection, even though in this snake it was very minor.
A second explanation mooted was that perhaps the snake itself had overheated.
Noting that the other two snakes remained alive, the overheating theory was thought possible, but unlikely and no changes were made.
That evening the *A. hawkei* was noticed to be in a tightly coiled position and fixed, as if in convulsions. Feeling that death was imminent, the snake was placed in the freezer. The snake was not observed while in this state, rather it was merely picked up and euthanazed.
In hindsight the snake should have been observed for some time as this may have also given further indicators as to cause of death.
The snake had been at the cool end of the cage and hence it was again determined that the Death Adders would not allow themselves to overheat and go to the cooler parts of the cage if necessary.
The *A. antarcticus* in the room remained alive and unlike the other two snakes had not been restless and so it was left unchanged.
In line with 'healthy' Death Adders this snake was effectively thermoregulating by straddling the hot part of the cage to maintain it's preferred temperature.
By this stage, certain facts became evident.
The snakes appeared to be dying of the same complaint or consequences from it.
Respiratory infection appeared to be a common denominator, with the possible exception of the first dead *A. hawkei.*
All snakes had been restless in the day or so before death, which is totally abnormal behavior for Death Adders. In captivity, these snakes usually just "sit", except when adult males go looking for a mate, which clearly wasn't the case here.
Other movement is literally from "A" to "B" and not in the genre of exploring or hunting.
Noting that the cages were literally sealed from one another, except via air, it appeared that the ailment must have been airborne, although the feeding implements had also been suspected as a potential means of transmission.
At this stage, there was no definitely known cause of death, but a virus appeared to be the likely culprit.
Another variable of note was the sloughing cycle. Both snakes to have died on 15 June 2003 were at the terminal phase of the sloughing cycle. Both snake's were in the situation where their eyes had been cloudy and cleared for more than a day and they were literally about to slough.
It seemed that this may have been a danger point for affected snakes and/or that affected snakes that slough are more likely to survive.
The *A. antarcticus* (AAA-106) in the same room was also heading towards a slough (estimated to be due about a week later) and therefore came under close observation, in anticipation of death immediately prior to slough.
Noting there were now three deaths that could not be satisfactorily explained and a possibility that these events could be repeated, it was decided to have the snakes examined by a pathologist.
This was arranged via a friend who had an account at a pathology lab, but the examination couldn't be done for a week.
Based on considerations of cost and the (in hindsight erroneous) assumption that no further deaths were imminent, the delay in examination was accepted.
On 15 June 2003 AAA-106 was noticed 'digging' with it's head. Based on close observation, this was seen to be a case of the snake wiping fluid and encrusted fluid from the infalabials.
As a result the six adult-sized Death Adders in the other room were inspected and three showed evidence of similar digging. One of the trio also had partially blocked nostrils as evidenced by it's breathing and popping of blockages.
On 21 June 2003 the two apparently recovered Death Adders were assist fed small mice.
On 22 June 2003 the apparently recovered *A. cummingi* (AC-3) regurgitated the mouse. The *A. antarcticus* of the same size did not regurgitate the same food (which was digested without incident).
Also (casually) noticed at this time was the newly restless nature of the *A. cummingi* in terms of wandering around the cage.
This snake was then transferred to the warmer (23°C) room.
Five hours later this snake was assist fed a small fish. The theory being that the small fish would be more easily digested and the snake would then have a positive calorie balance and the benefits that brought.
It was (wrongly) assumed that once the snake had eaten the fish, it would do what normal snakes do and that is find a nice warm spot to sit in and digest it's meal.
The following morning the snake was again observed wandering around the cage aimlessly, but it had not regurgitated the fish. As indicated already, this was clearly abnormal behaviour as snakes that have just eaten as a matter of course find a warm place and "sit" in order to digest their food.
A few minutes later I heard rustling in the cage. I rushed to have a look.
The snake was in a loose S-shape and rolling over in the cage in a "death roll". Its mouth was being held wide open. After about ten seconds the snake stopped in an unnatural position and it's body had nervous twitches, not unlike a snake that'd been just run over.
The snake was picked up and felt to be relatively cool (around 23°C) and it was noticed to still be breathing normally. Through the ventral scales, the heart was seen to be beating normally.
There was no evidence of attempted regurgitation of the small fish that remained in the stomach.
The snake was then euthanazed by freezing.
This snake had sloughed three weeks earlier, thereby negating the theory that the sloughing cycle is generally indicative of the timing of death.
Furthermore, on the evening of 23 June 2003 AAA-106 sloughed without incident.
This was expected.
The reason was because unlike the two sloughing snakes that died a week earlier, this one had not been abnormally restless in the days prior.
As per normal healthy snakes, this one simply stayed put, sloughed and then stayed put again.
Notwithstanding this, and the fact that the nostrils were totally blocked prior to sloughing, this snake retained a respiratory complaint as evidenced by 'popping' when breathing, after sloughing.
The most recent death and observations of this and the other snakes prior to death confirmed several important facts and effectively eliminated a number of possible causes of death.
This death of this latest snake was not caused be respiratory failure or blockage. Instead it appeared to be something nervous.
Bacterial cause of death (or complications thereof) was therefore unlikely, especially noting the minor nature of respiratory complaint in this snake.
In other words, for this snake a septicemia-based death, built on a systemic infection appeared extremely unlikely. Hence my looking for another cause of death.
Bearing in mind that this was the first death observed, save for similar observations in the second *A. hawkei*, it became likely that the other snakes may have died by similar means.
Noting this latest unexpected death (sharp decline in 48 hours), a new urgency appeared in terms of accurately diagnosing the problem before there were more losses.
Attention then turned towards airborne viruses (as thought to be the cause).
It was at the time of the death of the fourth snake that for the first time I was able to effectively piece together the pieces of the jigsaw

as seen and relate it to OPMV as documented in the literature.
Miscellaneous behavioral traits prior to death that had been noticed and yet more-or-less ignored in terms of significance or pattern, such as the wandering in the period before death, not thermoregulating in the same manner as 'normal' snakes, the neurological signs in the week or two prior to death and so on, were for the first time re-evaluated in terms of known OPMV symptoms.
By way of partial explanation, in the week or two prior to death, all the snakes to have died had been seen resting in unnatural positions, such as that seen in pythons with IBD. This seemed to be a precursor for the decline to death.
Snakes that died had deteriorated within a fortnight or less from outwardly healthy, feeding and well-fed individuals to relatively emaciated.
Working back through the records, it was evident that all but one of the eight small Death Adders had shown symptoms of this virus. Three had apparently recovered or were well on the road to recovery. One snake that died, the fourth one had apparently nearly made a full recovery only to rapidly relapse and die.
Other notes of relevance included that loss appetite was one of the first steps in the rapid decline in health. This appetite loss was measured in terms of snakes that would voluntarily feed now refusing to do so.
My feeding records had masked this factor as those snakes were simply assist or force-fed.
Notwithstanding this, those declining snakes would assist feed or be force fed and digest food at all stages of the infection other than at the terminal restless phase in the day or two preceding death. As a result, most snakes in my care tended to retain condition as the OPMV infection progressed. This would probably go against the trend in other collections where non-feeding snakes would probably be left unfed and presumed to have self-starved to death.
Records showed that almost any food assist or force-fed to the snakes at the terminal phase would be regurgitated, perhaps in response to the failure of the snake to literally sit still and digest it's food.
Another universal indicator of decline was the deliberate seeking of cooler parts of the cage by the ill snakes. The affected snakes did not thermoregulate as did healthy ones.
Noting the positioning of the cages and the relative sizes and robustness of the snakes affected, there was initially nothing likely that would indicate those snakes likely to be affected as opposed to those that wouldn't, at least in terms of snakes of the same general size class (as in four snakes from a litter of four month-old snakes). Nor was there any indication as to those which would survive the infection and those that wouldn't.
Having said this, there was a noticeable trend among infected snakes that did apparently determine survivorship.
Those that exhibited respiratory symptoms only appeared to survive. By contrast, those that also showed nervous disorders in terms of unnatural resting positions died.
This effectively meant that in the case of these snakes, death could be predicted up to a week in advance if the first signs were detected.
Another trend of note in these cases was that the snakes that were being aggressively force and assist fed tended to survive. The only possible exception to this was one of the *Acanthophis cummingi* although it too was left unfed for a period after it initially went of it's food.
The baby Death Adders in my collection came from three separate facilities. At those facilities and others that received the off-spring there were no die offs like experienced above.
This effectively cleared them as the source of the original infection. Noting that the pythons in the room were apparently unaffected and had been held since 2001, it was evident that the virus was A/ Not from them and 2/ Even if infecting them, was leaving them unscathed and effectively targeting the Death Adders.

THE DIAGNOSIS

The eventual diagnosis of OPMV by myself on the morning of 23 June 2003, arose on the basis of a process of elimination.
Significant here and of major importance for other keepers is that the diagnosis was made on the basis of clinical signs and an accurate appraisal of the housing and husbandry of the snakes only. There was no histological or microscopic examination prior to the diagnosis. This is important as it means that diagnosis can be made earlier than previously thought and due to the highly infectious nature of the disease, potential casualties reduced.
Possible causes of the deaths in my snakes that had been mooted, including those above, also included exposure to pest-strip and a delayed reaction (dichlorvos). This failed on the basis that all young Death Adders suffered the disease at more-or-less the same time, whereas based on the idea that the pest-strips were the cause, the two *A. hawkei* shouldn't have got sick for some months.
Furthermore past usage of pest strips to treat mites (including preemptively for incoming snakes), indicated that the dosage levels used for these snakes was well within safe limits (small section of strip (2.5 cm square) with snake in 30 cm long container for 30-60 minutes only).
It's also worth noting that in the past I experimented with massive amounts of pest strip exposure on reptiles, including a full strip (15 cm X 9 cm) with a snake and a lizard overnight in a small container and neither showed any adverse affects.
That respiratory failure caused death in all the snakes and was the root cause was refuted by the last death.
Fluid transferred diseases were initially ruled out on the basis of the physical isolation of the snakes and the lack of mites or other means of transfer (see below).
Husbandry related ailments were comprehensively rejected on the basis of past success with the same snakes (*Acanthophis*) in identical conditions, including the five adults raised the previous year.
A perusal of all available literature in terms of reptile diseases in general, including the major works of Mader (1996) left no alternatives other than OPMV as the cause of death.
Furthermore the piecing together of the known information and the observations of the snakes that died, also revealed a pattern typical of OPMV as detailed by Jacobson (2000, 2003a) and others who have published extensively on this specific ailment.
In fact that pattern of infection and death was textbook OPMV.
There simply was no other alternative!
In the morning of 23 June 2003 and with a new sense of urgency, confirmation of the diagnosis of this disease was sought. This was not as easy as expected. Being a Sunday, most of my calls were simply carried over to the next day.
On the Monday, the pathologists who were to do the inspection said that frozen corpses (as I had) were not suitable for histopathology. This was also confirmed over the phone by veterinary surgeon Peter Cameron, who suggested euthanazing another seriously ill snake, if and when that became available.
It was evident that by this stage, the only way to confirm diagnosis of OPMV in the dead snakes was via Electron Microscopy (EM) and so I made phone enquiries with this regard. Herpetologist Neil Davie was able to direct me to Gary Crameri of the Australian National Animal Health Laboratory (ANAHL) for these tests.
On the same day (24 June 2003) the corpse of AC-3 was forwarded to ANAHL in Geelong for confirmation of the diagnosis via electron-microscopy.

OTHER PREVIOUSLY IDENTIFIED AUSTRALIAN CASES

As of mid 2003 and prior to the spread of infection as connected to the cases detailed in this paper, the following appeared to be the (known) situation in Australia.
According to the records of Queensland-based reptile vet, Clayton Knight, of Deception Bay Veterinary Clinic presumptive diagnosis of OPMV has been made in Australia for 'about 8 years'.
Based on specimens he's seen and the histological examinations of them, he's noted a greater preponderance of neurological symptoms as opposed to respiratory in terms of causing death, even though respiratory disease appears in most suspected cases.
This more-or-less equates with the deaths I experienced.
His own view is that IBD is rare in Australia by comparison.
In Melbourne, specialist reptile veterinary surgeon Peter Cameron, of North Altona Veterinary Clinic has made presumptive diagnosis of about half a dozen cases and just one of IBD.
Cameron formed the view that OPMV was now fairly common in Australian collections but that it is underdiagnosed due to several factors including reluctance of keepers to pay for histological examinations of reptiles that are already dead.
In his broadcast e-mail of 25 January 2003, (Weigel 2003a), John Weigel said much the same thing in an e-mail which read in part as follows

Mauricio,

I am surprised that you didn't give me a 'heads

up' prior to widely distributing the NSW reports detailing the probable presence of OPMV at the Australian Reptile Park. Your broadcast email was forwarded to me by Peter Mirschin. I have been working with NSW Dept Agriculture on the matter of suspected paramyxovirus in a part of our collection since mid-November, and was told that I would be kept in the loop. May I ask who provided the reports to you? I have tried to contact you on your telephone numbers today, but without success. Please note that the many cc's for my (present) message were lifted from your cc list….

we have only had to inform very few collections of the need to use caution re snakes we have supplied…

We have reason to believe that the OPMV or something similar is widespread in collections, and ARP is merely the first collection that effectively sought correct diagnosis and/or did the responsible thing and immediately reported the diagnosis to relevant disease control authorities. Taking the optimistic view on human nature, OPMV is easily missed in infected snakes due to the fact that the immediate cause of death is most often a bacterial pneumonia or other bacterial infection.

…

Whether or not this is so, can probably never be determined, but notwithstanding the comments (preceding) there is some strong evidence to suggest that OPMV is in fact new here in Australia (see later).

Both vets named above said that most of their cases of suspected OPMV related to pythons, but that was expected as these are by far the more common snakes in captivity in Australia.

Notwithstanding this preponderance of cases relating to pythons, the experiences of both the Australian Reptile Park in 2002 and myself in 2003, indicates that other species such as Death Adders (*Acanthophis*) are far more susceptible to OPMV-type infections than most other reptiles. This trend is also evident in research results from the USA, which rates elapids as being more susceptible than pythons.

According to Nichols, et. al. 1998:

'The relative degree of susceptibility of snakes to paramyxoviral disease is Viperidae>Elapidae>Colubridae>Boidae'

The fact that elapids are not rated as highly susceptible as viperids may in part relate to the lesser number that are captive in the USA and Europe rather than the real position in terms of susceptibility.

As to other reasons as to why pythons feature in suspected OPMV cases that veterinary surgeons get and not elapids, there are other important factors.

As a generalization, it is only inexperienced reptile keepers with one or two pets, who take sick reptiles to veterinary surgeons. On the basis of costs and numbers more experienced keepers with large collections tend to source drugs and treatments themselves and self-treat their reptiles.

And it's only the experienced keepers who tend to have elapids!

Working backwards, some other mass die outs of Death Adders and other elapids in various collections attributed to poor husbandry in the past may in fact have been due to undiagnosed OPMV.

Speaking with various keepers in the Eastern states who had experienced numerous undiagnosed deaths, it appeared at first that OPMV may be an underlying cause.

However in many of these cases, OPMV was able to be eliminated as the cause as more detail became available.

OPMV remains little known, particularly in terms of different viruses or strains and the effects on different species.

In terms of keeping OPMV out of collections, Jacobson et. al. 1992 as paraphrased by Oros, et. al. said:

'Because there is only effective treatment against secondary bacterial infections, strict quarantine of newly acquired snakes should be followed to prevent the infection from spreading.'

In terms of the neurological effects and likely death via these there is no known treatment or cure.

Strict quarantining of incoming reptiles, implements and the like would including separate housing of incoming specimens for at least 6 months (excluding periods of dormancy).

However the 'expiry date' of the virus in terms of snakes or collections that have survived infection and apparently become immune has not actually been determined.

And it is this premature introduction of an infected snake into my mainstream collection that caused my die off!

MEANS OF TRANSMISSION

Before the publication of this paper, the most recent views on the subject were stated by Elliott Jacobson on his website at:
http://www.vetmed.ufl.edu/sacs/wildlife/Pmyx.html
It read:

'Transmission most likely occurs by virus being expelled into the air as droplets from the respiratory system. Virus gaining access to water bowls and pools of water may persist for considerable periods of time. Transmission of virus via the digestive tract through feces is also a possibility. Although transovarian or transuterine transmission has not been firmly established, this may also be involved in the spread of the virus.'

Until now, the general view among veterinary surgeons and private herpetologists has been that the virus is transmitted by airborne means in situ.

I can now report that this is not so (the evidence of this follows in this paper). While airborne transmission may technically be possible, it is not as a rule the case.

In practical terms transmission is by one means only: Fluids.

This includes blood, including as transmitted via snake mites, saliva, including as transferred via water bowls, shared water bowls (in which the virus can persist for some time), saliva left on food tongs and the like.

Noting that saliva and other fluids from snakes can be smeared onto a person's hands and remain moist for some time, it is even remotely possible that handling of reptiles by keepers in one another's collections may spread the virus.

Jacobson also wrote:

'The natural host for OPMV is unknown.'

However it's likely to be a species of snake that can harbor the virus without ill effect.

TRACKING BACK THE HOSER INFECTION - THE EARLY PART

It took about a week after the death of the fourth death adder on 23 June 2003 to get a complete and clear picture in terms of the Hoser OPMV infection, its transmission within the collection and related issues.

Notwithstanding this, the tracing of the infection through other collections based on the accurate diagnosis of my own source was remarkably easy.

The delay of some days in terms of getting the full picture was due to a masking of things in my collection as shown shortly and due in part to the rapid movement of infected snakes between collections.

Based on the above, the incoming adult *A. cummingi* or the *P. porphyriacus* were initially suspected to have been the most likely sources of the infection.

Both Eipper and Rossignolli who received the *P. porphyriacus* reported that some of their snakes died from OPMV-like symptoms (as described above in terms of appetite loss and/or respiratory complaint). By end June 2003, Eipper had lost five out of six juveniles, (the sixth dying a few days later) and four adults survived. Rossignolli lost one out of four adults, of which he later said, 'it just stopped eating and starved to death'.

Notwithstanding this indicator that the *P. porphyriacus* had OPMV, and noting what was published about the OPMV virus being airborne it was thought possible that the OPMV in the *P. porphyriacus* may have come from my snakes, not the other way around (see later).

None of the dead *P. porphyriacus* were postmortemed or tested for cause of death.

(Such failures are part of the reason that diseases such as OPMV may run rampant in the herpetological community).

The first *A. hawkei* death occurred within a week of obtaining it and as such is regarded as aberrant. The snake wasn't autopsied (partly on account of it's small size) and unlike the latter three snakes that died, there remains that the chance that it's death may not have

been from OPMV and was from some other as yet unknown cause. The corpse is retained.
In terms of the incoming adult *A. cummingi* not much was known initially, other than the fact that some Taipans (*Oxyuranus*) that Bigmore had passed on to Rossignolli at the same time also exhibited signs of respiratory complaint when obtained. They were all large adults and apparently recovered.
Checking through my notes, in particular noting the respiratory problem in the large female *A. cummingi* and the death of the adult male and that AC-4 had shown signs of OPMV as far back as April 2003 in terms of trouble sloughing and later respiratory complaint, it was assumed that the female *A. cummingi* had brought the infection into my collection.
I then (erroneously) assumed that the virus was transmitted via airborne means to the *P. porphyriacus.*
A scenario thought too horrifying to consider was that both the *A. cummingi* from Bigmore and the *P. porphyriacus* had OPMV.
As it happened, that was to be the case!

TRACKING BACK THE HOSER INFECTION - THE LATER PART

That I had an OPMV infection in my collection was known. At least from 23 June 2003!
As Scott Eipper had previously advised me in phone conversations of his dying *P. porphyriacus* my phone call to him on 23 June 2003 was merely to confirm a potential diagnosis of OPMV by myself in terms of his snakes.
What had previously been told of as cases of death by 'unknown causes' became subject of some questioning by myself.
The result was a summary of death via a process of rapid emaciation, dehydration, respiratory complaint, improper thermoregulation, wandering in cage and then being found dead for each and every snake … all of which fitted OPMV.
This was particularly so noting the general hardiness of the species. In other words he had the same problem as myself.
Ditto for Rossignolli's dead *P. porphyriacus* although his recollections were not quite as detailed.
In terms of OPMV and noting it's high degree of contagion, it was imperative that myself, Stuart Bigmore, Eipper, Rossignolli and others of relevance knew the state of play.
All were phoned and then e-mailed all relevant information as taken from the internet (via the search engines and so on), including a general precis of what was known about the virus.
In other words, these people were being advised of the fact that they either had the virus or probably did and to quarantine their own snakes from all others, including each other and also including other collections.
Since receiving the infected *P. porphyriacus*, Eipper alone had transferred snakes to six other people! All were contacted immediately and along that line at least, the infection was stopped.
I called Bob Gleeson on Monday 24 June 2003 to tell him that *P. porphyriacus* sent from him and been provisionally diagnosed with OPMV and were dying.
Overlooked at the time (but for a few days only) was another transaction of significance.
In early May 2003, Fred Rossignolli shipped six X one month old Tiger Snakes (*Notechis scutatus*) to Gleeson. Another six went to another NSW-based keeper Alex Stasweski. When picked up by myself to take from Fred's residence in Victoria to NSW the snakes had mites.
These mites were removed immediately by myself using pest strips and besides that the snakes presented as healthy.
The snakes started dying about a month after the keepers got them, so that by end June 2003, Gleeson was down to three and Stasweski was down to two.
Both described OPMV type symptoms prior to death.
Three weeks after dropping off the Tiger Snakes at Gleeson's I took the *P. porphyriacus* to deliver to Melbourne.
They too had mites, which were found to be ubiquitous in Gleeson's collection.
The mites were removed with pest strips in Melbourne before being passed on to Eipper and Rossignolli.
As already noted, they had OPMV.
For Rossignolli, this was the second time he'd brought in OPMV infected snakes!
For Stasweski, the problem was particularly serious as he also had a litter of baby *A. antarcticus* in the same room!
Unwittingly, I'd been transporting a plague of death from Victoria to NSW and back again!
In terms of the month-old Tiger Snakes, another Melbourne-based keeper took the balance of the litter the day after I took 12 to NSW, hence he too got OPMV into his collection.
Interestingly however, Eipper obtained a pair from Rossignolli the day after they were born. (Snakes born on 27 March, taken by Eipper on 28 March).
At the time, they were mite free and as of end June 2003 were evidently OPMV free and doing well.
And so in terms of the OPMV infection in my collection and that to which I had a role, the above sums up the state of play preceding the diagnosis of infection.
It seems like a pretty shocking record, but I think is actually far better than it could have been.
In my case it was merely days after the first death that I was onto the cause and alerting relevant people of the state of play.
Weigel and the ARP took 8 months.
Now if that seems bad, it's worth noting that no other keepers in these chains (running to Weigel) and beyond, or to myself, previous and beyond, even got to the point of diagnosing the virus and therefore never advised of the virus to people they'd passed snakes onto (until either myself or Weigel (allegedly) sent the message out).
The point I make is simple: Private reptile keepers have until now been too casual about how they deal with death and disease in their collections and the consequences as seen here can be dire!
There was another issue of note. That was from where the Bigmore infection came from.
As I had no idea where his collection of about twenty snakes came from I could only guess and so didn't pursue this line of inquiry.
He possessed foreign (exotic) snakes as in Boa Constrictors (*Boa constrictor*), Burmese Pythons (*Python molurus*) and Retics (*Python reticulatus*).
I initially assumed that the infection may have come in via these snakes, but was unhappy with this theory on the basis that those snakes had been held for some years and the evidence all indicated that the OPMV was recent to Bigmore's collection.

FACTS, QUESTIONS AND ANSWERS ABOUT OPMV AND THESE CASES

The published literature on OPMV has indicated certain inalienable facts.

- OPMV is a virus.
- In the laboratory infected snakes progress in a more-or-less linear manner, until death, or if they survive, tend to develop symptoms within a well-defined time frame.
- Contrary to the above, in reptile collections, OPMV outbreaks may or may not occur simultaneously.
- In the laboratory symptoms usually occur shortly after infection and death is typical within 3 weeks.
- In the laboratory it took up to 6-8 weeks for affected snakes to produce detectable antibodies to OPMV.
- Anecdotal reports, including in the scientific literature implicate snake mites as being a vector for OPMV.

In terms of my own outbreak and determining it's spread (retrospectively) I had the benefit of detailed records and hence was able to make a number of previously unreported findings.
But the questions of note tended to stem from the erroneous assumption that the infection was airborne.
They also arose on the basis of the established fact that my own cases of OPMV could have their infection dates determined on the basis of first signs of symptoms and death. In other words the deaths were within 8 weeks of infection and before the development of antibodies and recovery.
These known facts also explained why the large female *A. cummingi* took several weeks to recover from the apparently intractable respiratory infection.
The initially asked questions included:

- Assuming that the OPMV came into my collection on the adult *A. cummingi*, why did it take so long for an airborne infection to affect the rest of the collection?
- Was it possible that there was another non-airborne means of infection and if so what?
- If OPMV is airborne, why are mites implicated with

it's spread by some authors. Surely airborne viruses don't need mites as a vector?

- The baby *A. cummingi* were in cages in a line marked 1, 2, 3, 4. and, based on symptoms and/or deaths were infected in the following order 4, 1, 3, with 2 being missed. Why?
- Noted by myself, Alex Stasweski and Rob Gleeson was that OPMV infected snakes stopped growing. In my case this was reflected in the growth records of all affected snakes as in those who died and those who recovered. Noting that one of the young female *A. cummingi* didn't show any symptoms or stop growing, it was assumed it hadn't been infected. The question again is why? This is especially as the snakes on either side were.
- Was there a way to determine which snakes would live and which would die?
- What is the 'use-by date' in the virus in an infected snake that has recovered?

All the above questions, except the last, can in fact be easily answered based on my own records and the answers to go to the heart of dealing with OPMV in infected collections.

That OPMV is not usually airborne is proven by the fact that the large *A. cummingi* failed to transmit the infection to any other snakes until after the same feeding implements were used for this and the other snakes.

Noting that water bowls in cages were separate and not cleaned with a shared cloth, the only means of transmission was the feeding forceps.

In my case these are about 30 cm long and are used to hold mice and the like in front of feeding snakes.

In the case of my snakes, the infected adult *A. cummingi* obviously bit the forceps (actually many times) and the saliva left behind was the infected fluid that was then wiped onto other rodents which were eaten by other snakes.

In terms of the smaller assist-fed snakes, the ends of the forceps were then wiped inside the mouths of uninfected snakes.

This also explained why the allegedly airborne virus was unable to jump from the cages of the infected young *A. cummingi* into the uninfected cage.

Thus to stop the spread of the virus all that I needed to do was to stop using infected forceps.

All other aspects of my husbandry acted to prevent the natural spread of OPMV in my collection.

A lack of mites in my collection also prevented spread of OPMV from one snake to another.

That OPMV is in the normal course of events spread via fluids and not air, was further demonstrated in the case of Rossignolli.

Newborn Tiger Snakes (*Notechis*) that had been placed into a new cage separate from the rest of Rossignolli's collection were in the first instance unaffected.

Noting that he had the virus in other snakes in the same room, transfer would have been a given if it were airborne. Instead it was only after mites had made the move into the cage that infection took place.

The transfer at Gleeson's facility from Tiger to Black snakes was also a function of the mite infection.

As of end June 2003, Alex Stasweski had no mites in his collection and was using separate caging and implements for his Tiger Snakes. Four of six died, but so far there was no evidence of cross-infection of immediately adjacent cages with young and highly susceptible Death Adders.

Then there are some other assumptions that could be tested on the basis of my own records and those connected to this OPMV outbreak.

Assuming for a moment that OPMV has been in Australian collections for some years (see below), a logical question facing myself was why I never had an OPMV infection over many years of maintaining a huge collection in the 1970's and 1980's.

The only answer I can think of is that because almost all my reptiles had been caught by myself in the wild or were progeny of them, as opposed to being from other collections, there was no route of infection into my collection.

By contrast, none of the snakes mentioned in this paper were caught in the wild by myself.

Notwithstanding the above, the general view of most herpetologists, veterinary surgeons and the like is that OPMV is a recent import into Australia and this it was not present in the past.

Trying to go back into the past to test the assumption is quite difficult, but I can say with confidence that I have never come across anything like what's documented above and believe that it may well be a recent import.

There is also one sizeable piece of evidence in favor of this contention.

In the early 1990's Fred Rossignolli amassed a sizeable collection of venomous snakes for his snake-show business. These came from keepers all over Australia, including many who routinely trade in reptiles.

He has on and off continued to acquire new snakes to 2003, albeit on a reduced basis and as recently as September 2002 'borrowed' snakes from several prominent keepers to add to his large snake show that he does in an open pit at the Royal Melbourne Show. He'd done the same thing for some previous years shows as well.

The snakes are allowed to free-roam in the pit, intermingle and drink from a single water bowl for several days. Snakes are not removed at night. Mites are ubiquitous on Fred's snakes and that was the case in the 2002 show which I assisted with.

In other words, if there was OPMV in Victorian collections as of Sepember 2003, it'd be odds-on that Fred would have had it.

Noting the generally free trade between collectors in all Australian states, it's also likely that if OPMV were in collections in other states it'd have shown up in Victoria as well and including in Rossignolli's collection.

In 8 November 2002, Fred Rossignolli gave me on short-term loan an adult Female Death Adder (*Acanthophis antarcticus*). It was infested with mites and these were immediately removed via pest strip.

It was then placed for some days with two males in my collection in a single cage to allow them to mate.

As it happens, the males ignored the female and one (AAA-102) tried to mount the other (AAA-101).

They shared drinking water and any viruses would have transmitted between the snakes.

There was no transfer of OPMV or anything like it.

Feeding, growth and other records confirm this.

After sloughing and voluntarily feeding on 8 rodents in several feedings, the female snake was returned to Rossignolli on 10 January 2002.

In other words, we have established that as of November 2002, Rossignolli's collection did not have OPMV.

The infection into his collection must therefore have come from the Taipans he got from Bigmore on 15 February 2003.

THE SOURCE OF BIGMORE'S OPMV INFECTION

Stuart Bigmore of Lara Victoria, works for Ford Motor Company and in early 2003 took up an 18 month position in Japan. As a result his reptile collection was distributed to several other keepers to look after during his time of absence. This was in February 2003. All took the reptiles on an 'all care no responsibility' basis as a favor for him.

As a result several other keepers received infected snakes and infected their collections. Included among these was Haydn McPhie who took Bigmore's Scrub Pythons (*Australiasis amethistina*) and another keeper who took his King Brown Snakes (*Cannia australis*). 'Downline' from them in the following months there was little infection of other collections due to the limited number of snake transfers between collections.

However in February 2003, it was known by myself and Rossignolli that the snakes we personally received from him had presented in ill-health at the time, which is not normal for Bigmore in terms of his husbandry.

The room his reptiles are kept in is large and spotlessly clean. The building alone cost $40,000 to build in 2002 and is the envy of most other reptile keepers.

In terms of the spread of the OPMV infection in Bigmore's collection, that was easy to work out.

Water bowls in each cage were cleaned using the same cloth. Whatever cleansing agent he used didn't work to kill the virus. That was known from as far back as February, when two Death Adders from separate cages in opposite sides of the room presented with the same dysentery infection. This is typical of shared water.

Based on the above it was simple to assume that the OPMV I'd got, was present in all other snakes in his care, even if they did not all show symptoms.

The above is not an attack on Bigmore's husbandry practices, but

does show how even in the best looked after collections one simple oversight or procedural anomaly can lead to invasion by a well-placed infectious agent. A similar accusation could be leveled against myself in terms of the typical use of the same forceps to feed all snakes in my collection.

Taking charge of Bigmore's affairs in terms of his reptiles in his absence was Neil Davie, who incidentally was pivotal in organizing the EM on my dead *A. cummingi.*

Davie also confirmed my claim that the OPMV had come from Bigmore's collection when he noted that my claim explained the previously unexplainable fact that at Bigmore's facility an adult Scrub Python (*Australiasis amethistina*) had an intractable respiratory infection for some months that hadn't responded to Baytril (as they usually do) and that an Inland Taipan (*Oxyuranus microlepidotus*) had died.

Following the e-mailing to Davie of what I'd gained from the internet on OPMV, he phoned me to advise the following:

Bigmore received a Taipan (*Oxyuranus*) from Weigel in October 2002 and at no time before, during or since, had Bigmore been advised of an OPMV epidemic at Weigel's Australian Reptile Park. I was shocked to hear this and sought confirmation only to be told the same by both Bigmore and Davie (Bigmore 2003).

On 27 June 2003, Bigmore wrote:

> *'Raymond,*
> *I got the Taipan around October of last year from Weigel.*
> *I have never been contacted regarding OPMV.'*

The October date fitted well within the known parameters of how OPMV works in collections and explained it's relatively recent appearance in Bigmore's collection. In other words my suspicion that the OPMV source was not the long-term captive exotics was effectively vindicated.

The Taipan was further indicted by the following facts:

The ARP arranged a swap for a large Taipan of Bigmore's for a smaller one (the infected one). The ARP was to use Bigmore's for breeding with the pay-off that he'd get half the young.

Bigmore had not received any other new snakes or other reptiles in the 18-month period preceding February 2003.

This meant that the Taipan was indicted even in the absence of knowledge of an OPMV epidemic at the ARP!

The significance of this new information was completely staggering.

For Weigel not to have advised Bigmore of OPMV in October 2002 was totally understandable. The snake sent out was outwardly healthy and to that stage Weigel did not know the cause of his own die off, which by his own e-mail admission was literally out of control and spreading among snakes that previously seemed outwardly unaffected.

Weigel's e-mail makes it clear that quarantining of outwardly infected snakes failed to stop the spread of OPMV in his collection due to other outwardly unaffected snakes being carriers of the virus..

However when Weigel became aware of the virus and it's consequences in November 2003 he had an obligation to notify all persons likely to be affected and this included Bigmore, as Weigel himself indicated in his e-mail (quoted above).

Noting that Bigmore's collection wasn't distributed until February 2003, all OPMV cases derived from Bigmore's stock could have been avoided if Weigel had at any stage in the intervening three months advised Bigmore.

As to why Bigmore was not properly notified I can only guess (see below), but the toll of Weigel's known negligence and recklessness is huge and may have ramifications for many years to come.

It's notable that the first general knowledge of the Weigel/Australian Reptile Park outbreak was via a leaked e-mail that Weigel didn't approve of.

If Weigel had in fact alerted all recipients of his stock of the risks of OPMV, then perhaps his outrage at the e-mail may have been justified. But noting that at least one recipient of his stock didn't know of the OPMV epidemic, it's perhaps a travesty that the leaked e-mail didn't get a wider circulation.

In terms of myself, instead of archiving it, if I'd forwarded to those on my own e-mail list, including Bigmore, then perhaps the whole fiasco involving his snakes would have been avoided.

In a phone conversation on 3 July 2003, Weigel denied that his Taipan was the source of Bigmore's infection. Initially he denied sending Bigmore the snake. This later changed to him getting the collection manager at the ARP to call the courier, thereby somehow absolving him of blame.

Weigel then said that as the snake showed no symptoms of OPMV at the time it was shipped, it could not have been the source of Bigmore's outbreak.

Weigel said that as the snake showed no signs of OPMV it wasn't necessary for him to advise Bigmore of OPMV.

That contradicted the contents of his broadcast e-mail which made it clear that all recipients of ARP stock had been notified.

That all merely meant that the Taipan from the ARP was a carrier, nothing less.

Furthermore, based on the spread of OPMV in both Bigmore's and Rossignolli's collections we now know that Taipans (*Oxyuranus scutellatus*) generally survive OPMV without complication. In other words they can be asymptomatic carriers

The whole basis of the phone call was to demand and force a retraction of the idea that the ARP's Taipan was the source of the Bigmore OPMV infection.

However, Weigel provided nothing to rebut the already irrefutable evidence from both what we knew about Bigmore's collection, the spread of the OPMV from there and the details of Weigel's own broadcast e-mail, all of which proved emphatically that it was the Taipan from the ARP that was the source of the Bigmore infection.

Weigel's phone call merely presented a posible explanation s to why he didn't notify Bigmore of the OPMV infected Taipan (that he didn't think it was infected).

It also meant that either the phone comments to that effect or the e-mail from him were untrue.

As to which, I can only guess.

So what exactly was the death toll directly caused by Weigel's negligence in terms of failing to alert Bigmore of the OPMV in the Taipan?

That's a question that cannot be answered in full because as of June 2003, most of those people affected are only just starting to count the losses of their snakes. Almost on a daily basis casualties mount.

But in terms of keepers downline from Fred Rossignolli alone, we have more than a dozen deaths!

In terms of dead snakes, we have dozens, with probably dozens more likely to die and perhaps hundreds infected with OPMV … perhaps indefinitely.

If I hadn't put out the alarm at end June 2003, the death toll would have gone far higher!

In other words, there is absolutely no doubt at all that it was the ARP Taipan that was the source of Bigmore's OPMV infection. The only element of doubt now is what Weigel's state of mind was at the time the snake was sent, again after the OPMV infection was detected at the ARP and again at the time he sent out the broadcast e-mail claiming to have notified affected collectors.

However based on what's now known about Weigel's own outbreak the fact remains that Bigmore should have been warned of the possibility of infection from Weigel and he wasn't.

Even if as Weigel were to (unreasonably) contend, that it was not his Taipan that was the source of the Bigmore OPMV outbreak, the fact remains that if Bigmore had been warned by Weigel, the infection would not have progressed past his collection.

SOURCE OF THE ARP OPMV OUTBREAK

The Australian Reptile Park (ARP) appeared to have kept the OPMV outbreak they had under wraps. The first I heard about it was via the e-mail of 25 January 2003 from Weigel sent to: Mauricio.Perez-Ruiz@nt.gov.au and others.

I'll repeat the salient parts. It commenced thus:

> *Mauricio,*
> *I am surprised that you didn't give me a 'heads up' prior to widely distributing the NSW reports detailing the probable presence of OPMV at the Australian Reptile Park. Your broadcast email was forwarded to me by Peter Mirschin* (sic). *I have been working with NSW Dept Agriculture on the matter of suspected paramyxovirus in a part of our collection since mid-November, and was told that I would be kept in the loop. May I ask who provided the reports to you? I have tried to contact you on your telephone numbers today, but without success. Please note that the many cc's for my (present) message were lifted from your cc list.*

Why Weigel wanted to keep the outbreak under wraps is uncertain,

however other parts of the e-mail indicated that the outbreak was now under control and that:

> *Because we have not been in a position to distribute many snakes since the* (2000) *fire, we have only had to inform very few collections of the need to use caution re snakes we have supplied.*

Based on the Bigmore situation, we know that part of the e-mail to be factually inaccurate.

The urgency of the situation is perhaps better summed up in the report that apparently generated the Weigel e-mail.

That was a report by Bruce, M. Christie, the Chief Veterinary Officer of NSW. It was also posted on the www at: http://www.schlangenforum.de/modules/XForum/viewthread.php?tid=4981 by Viele Grüße Maik on 23 April 2003. It in part read:

> *The disease has not (officially) been previously reported in Australia.*
>
> *OPMV causes respiratory disease with wasting and death, and can cause "die-offs" in many species of snakes, including elapids, which includes all Australian venomous snakes.*

The statement appears to corroborate evidence to the effect that OPMV is a new and deadly arrival in terms of Australian reptiles.

Now obviously the ARP did not generate the disease in their labs and deliberately unleash it onto the Australian herpetological community, so the logical question then becomes from where did this virus come from?

Weigel's first e-mail alleged the infection came from multiple sources based on his educated opinion. This view was later amended (in June 2003) to three likely sources, whom he said all denied any possibility of being the source.

If that is in fact so, then the three keepers probably need to be censured as wholesale denying the possibility of a snake from a large collection being a potential vector of OPMV and without relevant tests is at best conjecture and at worst reckless.

Wigel's censure of those keepers is in my view justified.

However his own hypocrisy is evident when in spite of overwhelming evidence he denies the possibility that a snake the ARP sent was a vector for the virus.

Notwithstanding this, the Weigel e-mails point to OPMV being derived from other Australian collections.

Weigel also points to recent imports of exotic non-native reptiles in anticipation of an amnesty as being the potential original source of OPMV in Australia.

Here he may be close to the mark.

However there is another side to this that is also worth exploring.

The OPMV that has devastated the above collections, including the ARP has essentially targeted elapids and while usually only moderately adverse to large snakes, is generally deadly to smaller ones and juveniles.

Most exotic snakes brought into Australia have not been elapids and assuming them to be more-or-less immune to OPMV or at least less likely to be carriers than elapids, the most obvious source would probably be an elapid.

Hence my own view that the relatively small number of imported elapids are of greatest interest.

Oddly enough it was the ARP itself that recently acquired some large King Cobras (*Ophiophagus hannah*), which by virtue of their size would be likely to mask an OPMV infection, but still infect other reptiles.

We know this by noting how well larger elapids have coped with OPMV infections, including most of Rossignolli's 50-odd large elapids.

Suspicion in this direction is added by the fact that the ARP was the first place that OPMV appears to have surfaced (excluding the suspected, but unidentified cases listed above). Also the ARP's own internal quarantine failed to stop the spread of the infection until identified at least 8 months after it first manifested, indicating that the OPMV may have spread through their entire collection and merely masked by many snakes failing to show detectable symptoms.

Thus it is entirely possible that the ARP itself may have brought in OPMV as shown by the lack of OPMV in collections such as Rossignolli's as late as November 2002, and as a result of the failure to notify Bigmore of the infection, the ARP may have in effect foisted this virus into the Australian herpetological community, and perhaps ultimately into the wild here.

My theory (above) in terms of the source of the ARP's OPMV outbreak may be erroneous. But the situation is far too serious to be left in doubt.

All suspected vectors and dead snakes at the ARP can (I assume) be examined, including electron micrographed in order to help ascertain the true source of the infection.

The potential seriousness of OPMV means that such investigation should be done sooner, rather than later … that is if the pathway hasn't been left for too long already to be obscured in total.

EVIDENCE FOR FLUID BORNE OPMV TRANSMISSION IN COLLECTIONS

OPMV cases presented to veterinary surgeons to date have generally come from zoological collections and large private facilities. Means of infection was generally not detected, but rather assumed.

The microscopic or sub-microscopic size of virus particles has led to the assumption that transmission was by air, probably via droplets expelled via the nostrils of affected snakes. This theory has had several factors supporting it. These include the obvious 'popping' of nostrils in affected snakes as air is expelled and the fact that snakes in separate cages in collections become affected.

Fluid borne transmission of infections in collections is usually done by mites (or so it's been generally believed) and yet OPMV has spread in mite-free collections.

The air-borne transmission theory was also thought to be attributable to the Hoser infection in the first instance. However a close appraisal of infection records tended to work against this theory. This was especially after it became clear that three Death Adders (*Acanthophis*) that had showed no symptoms were in fact unaffected as deduced by their continued appetite, growth and lack of respiratory complaint. One of the trio, a young *A. cummingi* was obviously not affected as the other three from the litter showed all the most severe symptoms of the disease, save for one, which merely lacked neurological signs and recovered.

However in the first instance the main reason the airborne infection theory was questioned was because the original vector a large *A. cummingi* was kept in a cage immediately adjacent to other unaffected snakes for two months, with direct straight-line air contact of less than 6 cm and yet there was no transfer of infection.

Transfer from this affected snake then effected more or less simultaneously over an apparently random selection of snakes some months later.

Hence the investigation of a new variable, any new variable that led to this new transmission.

The only change was the dropping of individual feeding and handling implements for the carrier snakes.

As it had been noticed that all snakes tended to bite the forceps when feeding on held rodents, it became obvious that here was a fluid transfer means.

In another similar example, the four young *A. cummingi* were housed in cages in a linear manner, numbered 1, 2, 3, 4 and yet the infection progress was 4, 1, 3 with 2 being missed.

Holes connecting the cages gave a potential nostril to nostril distance of less than 4 cm. Furthermore, noting the rectangular shape of the containers and the propensity of all snakes to sit more or less parallel and 'in line' in response to the positioning of the heat mat running along the underneath of the cages, these snakes would have spent some weeks more or less permanently within 20 cm of one another.

In spite of this, it was evident that airborne particles from cages 1 and 3 failed to infect cage 2.

A similar situation existed in terms of the two uninfected adult *A. antarcticus* which sat literally within 6 cm of infected adults including via direct line of nostril contact via cage air holes for some weeks without becoming infected and were left in this position after the OPMV was detected (firstly on the incorrect assumption that they were infected and not showing symptoms and then on the accurate assumption that there was no serious risk of them being infected, even though they were unaffected, due to the newly commenced practice of not sharing feeding implements).

In all other OPMV infections sourced down from Bigmore a fluid-based means of transmission was identified.

When none was found, there was no OPMV transfer. This appeared to be the case in the mite free newborn Tiger Snakes (*Notechis*) that Eipper received in March 2003 from Rossignolli and again in terms of the failure of the OPMV to jump from later infected *Notechis* held by Stasweski to uninfected *Acanthophis* immediately adjacent.

Further evidence of the fluid-based transfer of OPMV came in the general infections of the Gleeson and Rossignolli collections that both had mites throughout. Likewise for the transfer in the Bigmore collection which had a wet cloth as a transfer medium.
This also reconciles a correlation noted by previous authors in relation to OPMV, that being that snake mites are a vector.
If OPMV was in fact airborne in transfer, then mites would seem to be a particularly inefficient means of transfer and effectively a non-issue. We know this isn't so.
The fluid borne mans of transfer also explains differential rates of infection in collections in terms of it reflecting cleaning water bowls with a given cloth as they get soiled and not as a block.
This also reconciles with statements by Jacobson that indicate that the virus does not last long in dry environments and yet persists for some time in water (liquid environments).
It also reconciles with the need to physically infect snakes with fluid based medium in the laboratory situation if one intends infecting snakes.
A remaining question then becomes when do infected snakes shed virus (become infective)?
In the laboratory situation infected snakes become increasingly infected as the virus replicates. Based on the progress of the infection through the various collections, in particular that of Gleeson, it's fair to assume that snakes can become infective carriers within three weeks of first becoming infected.
The real time may in fact be far shorter.

THE PREDICTABILITY OF OPMV TRANSMISSION, INFECTIONS AND DEATHS

By end June 2003 and after contacting the various people who had received OPMV infected snakes, the nature of OPMV in terms of Australian collections had become generally known to me.
One recipient of 7 infected neonate Tiger Snakes (*Notechis scutatus*) from Fred Rossignolli, was first contacted by myself on 1 July 2003, which was well after most other OPMV affected keepers had been spoken to.
Prior to then I had not been in contact with him.
Before allowing John Debendictine to tell about his snakes, I was able to inform him that he had received OPMV infected snakes and that most if not all had died.
I was also able to tell him that the pre-death symptoms included loss of appetite, stunted or no growth and that he probably found the snakes dead in a belly-up position.
On all counts I was correct.
Debendictine received the snakes from Rossignolli in early May 2003, the day after I took the infected snakes from the same litter to two Sydney-based keepers.
Debendictine had lost six out of seven snakes. He at that stage had no idea as to cause of death.
All had appeared normal at first and ate and grew apparently normally for a few weeks before the snakes manifested symptoms and started declining. His most recent death had been a week prior (around 24 June).
All six had not been seen dying, but had been found belly-up in their cages. There was no evidence seen of respiratory infection.
These were the same things observed by Stasweski and Gleeson with their neonate Tiger Snakes.
In other words the effects of OPMV on these snakes was the same in every case and therefore tended to be predictable for the species.
Notable is that a year earlier Debendictine had received some other mite-infested neonate Tiger Snakes from Rossignolli a month after they'd been born and had raised every one without problem.
Once again, the relatively new OPMV was indicated as the cause of death as all other factors were the same.
Debendictine's observations again drew attention to a previously neglected trend in snakes that had died from OPMV, at least in terms of previously published accounts. In the case of the Death Adders that actually died in my custody (as opposed to being euthanazed before death), namely one *A. hawkei*, one small *A. cummingi* and Bigmore's original male *A. cummingi*, all were found dead in a belly-up position.
In other words such could (in combination with other signs) could be treated as indicative of OPMV.
Notable however was that in Eipper's Red-bellied Black Snakes (*Pseudechis porphyriacus*), there was no belly-up position at death. Also unlike the Tiger Snakes, all the Red-bellied Black Snakes showed some sign of respiratory complaint before death.
Hence the use of belly-up death (or lack of it) or respiratory infection or lack of it, to definitively indicate OPMV should not be relied upon unless the species trend is known.

FURTHER EVIDENCE OF DATES OF OPMV INFECTION AND THE LACK OF OPMV IN AUSTRALIAN COLLECTIONS PRIOR TO THE ARP OUTBREAK

Notwithstanding the assertions in Weigel's posts that he believed OPMV derived from private collections outside of the ARP's, the fact remains no hard evidence has been provided to support this contention.
In an e-mail dated 26 June 2003, Weigel (Weigel 2003b) has refused to answer questions put to him in this regard via an earlier e-mail from myself.
We have no names, details of snakes allegedly received and identified as sources, or the like.
Assuming all transactions to be legal, there should be no impediment on this information being generally and publicly made known.
One can only ask why Weigel refuses to have his e-mail assertions of 25 January 2003 tested for veracity.
As this paper relies only on hard evidence to support contentions, the source of the ARP's OPMV outbreak cannot be pursued beyond what is already related.
In other words it remains unknown.
But I can review some key facts should be reviewed as known.
As recently as August/September 2002, Fred Rossignolli borrowed snakes from several prominent reptile keepers to place in a large pit as part of a two-week snake display at the Royal Melbourne Show.
Due to the ever-present mites that almost immediately infested all snakes and the sharing of a single water bowl, any infections such as OPMV would have transmitted then.
As already noted, as recently as November 2002 Rossignolli did not have OPMV, nor did he have it in early 2002 when that year's neonate Tiger Snakes were distributed by Simon Watharow to several Melbourne keepers including Debendictine. Working backwards, and looking at from whom Rossignolli borrowed snakes in August/September 2002, one of those collections from whom he borrowed snakes was Bigmore's, meaning that at the same time he too did not have OPMV, further pinpointing the October 2002 date of infection.
Bigmore raised the possibility that another snake the male Floodplain Death Adder (*Acanthophis cummingi*) later passed on to myself, may have been the source of the infection.
The basis of this possibility was that the snake was a 'poor-doer' ever since being acquired.
That came from Andrew Geddy.
The snake was ruled out as the vector to Bigmore's collection on the basis of it beaing acquired 18 months earlier in 2001.
If it had carried OPMV, then Rossignolli's collection would have had it in September/November 2002 and we know for a fact that he didn't.
Furthermore Geddy had directly supplied Rossignolli with snakes in the period preceding November 2002 and yet we know emphatically that Rossignolli's collection had not got OPMV. Hence that snake and Geddy's collection in total was ruled out as a potential source for the OPMV.
Also noted was that Geddy's own cleaning regimen (same cloth, all cages) wouldn't have stopped OPMV from attacking his entire collection.
The investigation of Geddy and his collection was done as a matter of procedural fairness to ensure that any and all suspected snakes, and not just the ARP's Taipan were checked and either indicted or cleared as being potential vectors of OPMV.
Rossignolli borrowed snakes in 2002 from more than six other keepers. Those keepers in turn had derived stock from over 30 in the previous 12 months alone including most of Australia's better known private keepers, many of whom also supplied the Australian Reptile Park after the mid 2000 fire killed most of their own stock.
Based on the results already provided, there was no OPMV in those collections.
Hence overwhelming support for the contention that the OPMV now seen is 'new' to Australia.
Weigel has not provided any details of the alleged source/s of the ARP outbreak, so his claims cannot yet be independently checked.
This is a serious deficiency in terms of identifying the true source of the OPMV infection now being seen in Australia.
This again points to the ARP as being the original source of the

Australian infection, or perhaps an original outside source, which either recently acquired an infected animal from overseas and/or had little if any contact with other prominent reptile keepers, being the direct conduit for the infection into Australia.
Most importantly however is strong evidence that this strain of OPMV at least is new to Australia.

OPMV INFECTED SNAKES - TREATMENT AND RESULTS

In terms of the cases detailed here, there was little if any coordinated plan of dealing with the infection until well after much of the damage had been done.
This in the main stemmed from total ignorance by the keepers in terms of the causes of the disease and deaths at the time they were occurring.
Excluding myself, the other relevant keepers by and large regarded all the deaths as unfortunate but not from treatable or avoidable conditions.
In hindsight this was completely wrong.
All deaths in reptile collections should be treated as avoidable unless proven otherwise (regardless of cause).
One exception to this was Bigmore who attacked the respiratory infections with Baytril, the drug of choice for such infections. Rossignolli also treated snakes with respiratory infections the same way.
In the case of most of the rest, the OPMV infections manifested in young snakes at great speed and death resulted so fast that treatment of any form simply wasn't considered.
In the case of the Red-bellied Black Snakes, Eipper noted that they declined in condition rapidly and then died, appearing emaciated. Respiratory infection was noted merely as an afterthought and not as being a root cause of death in Eipper's view.
In terms of the Tiger Snakes, both Stasweski and Gleeson failed to note evidence of respiratory infection at all, even after being questioned about it. They merely reported that the snakes would decline rapidly and die.
I was able to observe a Tiger Snake dying from OPMV at Gleeson's facility on 4 July 2003 and failed to see any signs of respiratory disease.
The snake did however exhibit other traits of OPMV infection in terms of lack of growth as compared to healthy snakes, emaciation, unnatural resting positions, failure to thermoregulate and at the terminal phase seizures and convulsions.
In terms of myself, at the first instance the respiratory infections were overlooked and only dealt with when it became plain that at least one snake, the male *A. hawkei*, was in terminal decline.
It was also the only snake I had die that appeared to be debilitated by the respiratory infection. For the others it appeared as being 'present', but also merely an inconvenience as opposed to life threatening.
In hindsight that may not have been the best approach.
Treatment for respiratory infection is therefore indicated for OPMV infected snakes.
Baytril is the drug of choice for affected reptiles. It's properties include necrosis at the site of injection and rapid absorption into the system, even if ingested orally.
Based on these salient facts and the small size of the affected snakes (most under 30 cm), I chose to administer the drug into the water in the cage.
The liquid is fairly tasteless, even when concentrated and when diluted at the a few drops per 50 ml (in line with the treatment indicated for poultry), the water's taste remains effectively unchanged. The snakes will still drink the water.
In the first instance Baytril was added to the water of the young female *A. antarcticus* and after a week of no ill effect, the Baytril treatment was widened to include all Death Adders in the collection, including those visibly unaffected.
In terms of the neonate Tiger Snakes held by Stasweski and Gleeson, there were other factors worth noting.
Part of the general trend in OPMV infected snakes is a loss of appetite, even at the early stages of active infection. This much appears to be a general trend.
Stasweski tended to offer food to the snakes and generally if they refused to eat, they were left alone. His death rate at 4 out of 6 by end June was higher than Gleeson's 3 out of 6 (with a fourth death on 4 July).
This figure is particularly notable, given that Gleeson's also at one stage had to contend with mites, which is certainly no mean feat in small elapids.
Gleeson by contrast to Stasweski followed earlier advice from me and force-fed the snakes. He said one of the three deaths was due to error on his part by him force-feeding too large an item to the snake and it dying there and then.
Assuming Gleeson's diagnosis to be correct, this meant that only two (later three) of his snakes actually died as a result of OPMV. Noting the survivorship trends in my own Death Adders this again tends to indicate there is benefit in force-feeding OPMV affected snakes that would otherwise not eat.
While there are general benefits in maintaining a positive calorie balance in ailing snakes, the exact reason for the apparent benefits of force-feeding in OPMV affected snakes isn't known. But the trend is clearly evident.
Further proof of this trend was seen in Gleeson's housing of the neonate Tiger Snakes.
As late as 4 July 2003 all were still being housed in a shared cage. Due to the shared water, it was obvious that all must have been in contact with the virus here as well as previously and therefore become infected.
Two of the snakes remained outwardly healthy and had been force-assist fed throughout. They had apparently recovered from infection and were by that stage on a strong growth trajectory.
This mirrored the situation in my own recovered neonate *A. cummingi* (AC-4) and the two neonate *A. antarcticus.*

MANAGING OPMV IN COLLECTIONS IN AUSTRALIA

The full extent of the OPMV infection from Bigmore's collection is not yet established. However it's likely that as a result of the alarm being put out in June 2003, the spread of the virus will be checked. Moving up the line and down again to other recipients of Weigel's snakes is not known.
He has not disclosed who these people are, so their management of potentially infected snakes, results and so on are totally unknown.
Giving Weigel the benefit of any doubts and assuming that he did in fact notify other recipients of infected snakes as alleged in his e-mail of 25 January 2003, we get to the next problem which is the source of Weigel's infection.
Weigel claimed that he thought the infection came from at last three other keepers all of whom he alleges denied giving him stock that is in any way tainted.
If those keepers, (whoever they are) have made such denials, they should be questioned further due to what's known about the transmission of OPMV and how easily it can be masked within a collection, including via asymptomatic carriers.
OPMV in Australia may well predate Weigel's own actions in dispersing the virus, even though in the case of Victoria and parts of NSW at least, Weigel appears to have given the virus it's first major beachhead.
Based on known OPMV attributed mortalities in the collections of myself, Eipper, Bigmore, Rossignolli, Gleeson, Stasweski and others there are certain facts that have become known.

- OPMV targets certain species more than others and other factors increase risk of dying including size and general health.
- Smaller snakes are more likely to succumb than larger ones and elapids in general seem susceptible. Small Death Adders in particular are vulnerable to OPMV.
- Frequent force-feeding of moderately affected snakes (but not items so large as to be regurgitated) appears to correlate with increased chances of recovery and is therefore recommended.
- Secondary bacterial infections, such as respiratory, should be treated as required, including use of relevant drugs as needed.
- Noting the fluid-borne nature of OPMV and how it spreads in collections, water in separate cages should never have contact, including via cleaning medium and if feeding tongs are bitten or come into contact with a feeding snake's mouth then they should be sterilized before re-use on another snake.
- There are anti-viral washes on the market, but these do fail and so the best form of sterilization recommended is to immerse the item in boiling water for at least ten minutes. In other words metal feeding

implements are recommended.

- Mites remain enemy number one and must under no circumstances be allowed into a collection.
- Incoming reptiles should be quarantined for at least 12 months before being allowed with other reptiles, except in exceptional circumstances and with all risks being fully understood. Quarantine includes no contact in terms of food, water, caging or implements.
- Based on the assumption that OPMV is, or will become ubiquitous in Australian reptile collections in coming years, juvenile snakes (elapids especially) will pose special problems.
- Neonates are best kept in complete isolation from other reptiles until of adult size, at which stage their vulnerability to OPMV is reduced. This again means totally separate feeding implements and the like. If hands come into contact with infected snakes they must not come into contact with other snakes until thoroughly cleaned, dried and then exposed to dry air for as long as possible. If in doubt, use thin disposable gloves.
- In the event that you suspect you have OPMV in your collection, it is essential that you work both up and downline from your collection in terms of movements and notify all other potential carriers of the virus. Failure to do so would constitute gross negligence.
- In the event that you diagnose OPMV in a snake in your collection or have been advised of the posibility, it is important to assume that all snakes may be infected and each must be effectively quarantined from one another. Do not just separate those who outwardly show signs of infection. Others may be asymptomatic carriers.

The central theme of this paper is failure. My own collection quarantine, I thought of as being 'best practice'. It failed to stop an unknown and undetected OPMV infection, even if it was only by something as small and insignificant as a pair of forceps! Most, if not all other collections mentioned had similar failings, as in they allowed OPMV into their facilities and without effective barrier.

Furthermore my own view is that these are by and large better managed collections than the average.

As a corollary to this, I spoke with managers of most of Australia's largest public and private reptile collections in June 2003 and found without exception that every single one of them was effectively unable to stop and deal with an unannounced OPMV infection via newly acquired reptiles, such as a single infected and asymptomatic snake!

Noting that OPMV is likely to become a more relevant issue for reptile keepers in Australia and elsewhere in future, the above lessons are salient to all.

SPECIFIC MANAGEMENT NEEDS IN TERMS OF OPMV IN COLLECTIONS

Noting the likelihood of an increase of OPMV in collections in coming years, reptile keepers can best manage OPMV by asking themselves the following simple question:

If a reptile that presents as healthy is obtained and yet unknowingly carries OPMV, may it infect the rest of your collection?

If the answer is either a maybe or a yes, then keeping protocols need to be altered to remove that possibility.

Noting that the histological and EM aspects of diagnosis of OPMV are time consuming, usually done after death of at least one reptile and often not conclusive in themselves (in terms of histological diagnosis), diagnosis of OPMV will tend to be presumptive in most cases.

Notwithstanding this shortcoming in terms of diagnosis, because OPMV is such a serious virus and because it can be so devastating in large collections and those with large numbers of small snakes, especially elapids, management of OPMV in situ is required and preferably before deaths start to mount.

If OPMV is suspected in a collection, all possible steps should be taken to ascertain the following:

- Likely source of infection
- Means of transmission in a collection
- Reptiles likely to be affected.
- Plan of treatment for all reptiles likely to be affected and means to isolate those believed unaffected.

Much of what's required has already been outlined, however some points are worth noting here.

If in doubt, the worst should be expected and in large collections this can become a logistical nightmare. As a matter of procedure all cages, feeding implements and the like should be isolated from one another so that there is no further transfer of pathogens.

Other sources of transfer such as shared cloths, mites or forceps must be eliminated.

If there is a shortage of implements such as forceps (likely), then these must be sterilized between uses with different reptiles.

The best means to do this is via immersion in continually boiling water for at least ten minutes (hence metal implements only).

Baytril or similar anti-respiratory treatment should be added to water bowls of likely to be affected snakes preemptively to prevent these infections.

As a matter of course, all reptiles should be maintained in optimal condition and be kept well-fed, especially smaller and growing snakes.

Force/assist feeding of neonate and small snakes that don't voluntarily feed is recommended.

In terms of clearing snakes and cages as being 'no longer infectious', this part of OPMV management is not yet known.

However the following is known. OPMV does not survive long in dry waterless environments and a cage that has been completely dried out, de-mited and left empty for more than two months can be presumed OPMV free.

In terms of OPMV affected reptiles, this is harder to ascertain. Noting my own failure in this regard and other people's findings, OPMV must be regarded as being in infected reptiles indefinitely, unless and until it is proven otherwise.

If an OPMV carrying reptile is found to be gravid, it should be allowed to produce eggs or young in a dry mite free cage and eggs or young should be removed immediately. Based on what happened in terms of Rossignolli's Tiger Snakes (above), this should allow for OPMV-free young.

Notwithstanding this, these snakes should be quarantined for at least six months for signs of OPMV after being born or hatching.

Management of OPMV affected snakes has already been covered in this paper.

TESTING FOR OPMV IN LIVING COLLECTIONS

A method for doing this is postulated below. I have not myself yet done it, but do regard it as feasible. (If considering such experimentation in Australia, it's probably wise to seek regulatory approval).

A snake formerly infected with OPMV but suspected as being OPMV-free based on it's renewed good health for at least six months, should be placed in a new cage and without water for at least a week, or if this period is deemed too long, then for as many days as possible.

The snake can then have a water bowl with fresh clean water added to the cage.

The idea here is that OPMV is known to persist in water for some time and assuming that the snake itself has developed antibodies to fight OPMV any residual OPMV in the snake or picked up when drinking should be eliminated by the snake itself.

After another week or so in the cage and when it has become evident that the snake has taken drinks from the water bowl this snake can be removed and another OPMV free snake added.

This second snake should be of a type known to be very susceptible to OPMV. Examples include newborn Death Adders (*Acanthophis*), newborn Red-bellied Blacks (*Pseudechis*) and newborn Tiger Snakes (*Notechis*).

Assuming the snake does not develop OPMV, it is then reasonable to infer that the original snake was also OPMV free.

WHY DISCLOSURE IS ESSENTIAL

On 22-23 June 2003, when speaking to several people about the OPMV in my collection, some advised me against widely publicizing the fact. The advice centered on an assumption that some people may 'shun' or 'vilify' me as a result.

Among those people who proffered this advice was a respected veterinary surgeon. Whilst the advice given to me was candid and with a view to looking after my best interests, this was ultimately rejected.

Part of this rejection was based on the obvious consequences of the attempts by Weigel to keep his own OPMV infection generally unknown.

The beginning of Weigel's broadcast e-mail dated 25 January 2003 condemned the person who leaked out the details of the Australian Reptile Park's infection.
While other parts of Weigel's e-mail generally ran on the theme that the OPMV outbreak was under control and that all relevant people had been notified, we know that not to be true. Bigmore at least wasn't and the list of others may be extensive.
Whether or not Weigel's failure to communicate to Bigmore was due to a deliberate intent on Weigel's part or simply oversight isn't known. Furthermore, Weigel's habit of making obviously false and untrue statements in e-mails (at least) is well-known (for example see his e-mail of 3 Feb 2001 re *Pailsus weigeli*) or those he posted to the same list about Indonesian Pythons and therefore any explanation from him in this regard must be regarded as questionable. For that matter it also casts some doubt in terms of his e-mail dated January 2003 re OPMV which has more-or-less been accepted here as accurate.
The results of Weigel's failure to properly and/or widely advise of his own OPMV outbreak have in hindsight been devastating.
All collections down from Bigmore's could have avoided the OPMV outbreaks had Weigel widely disclosed his epidemic so that Bigmore knew that his own collection was at risk, and/or if Weigel had notified Bigmore direct.
Consequently, in the case of myself I chose the opposite tack and that was to notify all relevant parties of my infection.
As no reptiles had left my collection, that may sound simple, but due to my role as courier in the *P. porphyriacus* and *Notechis* transfers between NSW and Victoria I felt obliged to notify all relevant parties. Notable is that there was no ill-feeling displayed towards me, even though I'd literally been the courier of death for those people's collections.
The same protocol was adopted by Bigmore, Rossgnolli, Eipper and other persons in the chains below Bigmore.
The result being that as of the last week of June 2003, this leg of the OPMV epidemic was hopefully stopped in it's tracks.
If a fellow keeper is small-minded enough to condemn myself or the others in the chain of infection for having OPMV in their collection, or allowing it in, then so be it.
I am not perfect and freely admit that my quarantine system as of early 2003 failed to allow the virus in. But more importantly, if disclosing my own imperfections helps stop others from making the same procedural errors in the light of this relatively newfound threat, I'll put up with any condemnation.
Thus the advice must be:

> IF YOU HAVE OPMV IN YOUR COLLECTION, OR SUSPECT IT THEN NOTIFY EVERY RELEVANT PERSON AS FAST AS POSSIBLE.

THE CONSERVATION ASPECTS OF OPMV IN AUSTRALIA

A question that nagged others and myself in terms of this OPMV outbreak and other alleged OPMV cases in the late 1990's and since is why has OPMV not surfaced earlier as a problem in Australian collections. Two possibilities are that it was either present in collections for many years and simply undiagnosed, or perhaps it has been brought in, perhaps with non-native reptiles, which appears to be thinking of many people in Australia (see previous).
This is a question that should be answered sooner rather than later and may take some government expenditure to do so.
If OPMV is non-native it may at some stage in future pose a serious threat to native reptiles in a similar manner to which the Chytrid fungus has annihilated many native frogs. In other words, OPMV infected reptiles must never be let loose.
Based on this worst case scenario and further noting the means of transmission of OPMV (fluid borne), it is essential and urgent that strictly controlled tests be done on the effects of OPMV if released into the wild.
Notwithstanding this risk, my own educated guess is that OPMV (or variants thereof) would not pose a serious threat to most wild reptiles. The disease is to an extent limiting in that infected animals die and by and large reptiles do not regularly interact. A counter to this would be OPMV infecting a common water source or similar causing a die-off in a single location.
In the captive situation, even when there are mass die off's it's usual for some to survive (for reasons not always clear). Assuming the same to be the case in the wild, this would mean the worst case scenario in the bush would probably be a population crash, which in most (but not all) cases would rectify itself after a period of time.
A related issue is that of so-called snake controllers. These are the people who release reptiles into the bush after they have removed them from suburban houses and the like.
Hoser (1995) talks extensively about the potential perils of releasing wildlife back into the wild.
OPMV infection can be added to the list.
The Victorian wildlife authority is currently reviewing it's procedures in terms of releasing snakes into the wild.
It may well be safer to simply prohibit the re-release of most snakes.
Another issue facing licenced snake catchers is the potential passing on of disease from captive snakes to wild snakes in transit.
In my own case, as a licenced snake removalist in Victoria some snakes removed from private properties have at times been held in the room with the Death Adders 'in transit'. These specimens have included snakes caught locally, late at night and brought to the house by members of the public.
These snakes must be kept well apart from captive snakes.
In hindsight, the anti-mite treatment of all incoming reptiles, including those 'in transit' will not necessarily be enough to guard against OPMV-type infections if they are held in proximity to the permanent collection unless other isolating mechanisms are used, including never using the same handling implements, water containers and the like.
In terms of what pet shops and private dealers can do in terms of potential OPMV infections, well, that's a major headache for which simple answers are simply not available. As it stands, a sizeable portion of reptile-dealing pet shops in Victoria at least are struggling to cope with simple issues like mite plagues and other basic husbandry issues (although there are some notable exceptions).
The speed at which OPMV (and perhaps similar) infections can cause mass die outs in captive collections is further reason to split captive breeding colonies of rare and endangered species.
Noting that in most states there is only one or two major public zoos, concentration of populations of Australia's endangered species in such a handful of facilities would border on the reckless.
Sound conservation, especially of reptiles, must therefore incorporate more facilities, which must therefore include private keepers.
I therefore recommend a conference between relevant stakeholders to work out a protocol to deal with the key research issues indicated above and proper conservation and legal issues that may arise from the scientific findings that emerge.
My own view is that it should be a criminal offence for a person to knowingly trade in infected reptiles and/or failure to notify a recipient if and when a suspected infection occurs.
Movement permits for reptiles have long been a bone of contention for reptile keepers in Australia. They are generally regarded as being a waste of time and paperwork.
Notwithstanding this, the prospect of OPMV and similar infections spreading in Australian collections means that accurate tracking of movements is essential to stop them.
While permits themselves may be unnecessary it is recommended by myself that there is a system in force whereby a central register is kept of all wildlife movements in captivity so that infections can be tracked as needed.
If the system is free of charge, including for interstate transfers (as in Victoria at the moment, but not in NSW or Queensland), then compliance will probably be higher.
Outside Australia the whole issue becomes more difficult to deal with given the far freer trade in wildlife, including across international boundaries.

SHOULD EXOTIC REPTILES BE ALLOWED INTO AUSTRALIA?

This question is beyond the scope of this paper. However assuming for a moment that OPMV came into Australia via the illegal trade, that in effect would support the contention that open legal, and quarantined imports are preferred to the current 'ban' which has driven trade underground.
It's also notable that a respected institution as in the ARP, against which no allegations of illegality are made and who have access to the best available veterinary surgeons, vast amounts of money and funds, has evidently provided the source of a much wider OPMV outbreak.
Hence, my own view that one set of rules should be enforced against all reptile holders regardless of their status in terms of public, private or whatever.
My view is that a total ban on any exotic reptiles anywhere in Australia is preferable, to letting them in to be traded legally. However for a

ban to be effective, there must be NO EXCEPTIONS.
The no exceptions should include major public zoos and significant private holdings as well.
Backed by immediate euthanazia of all exotics if located by officials - no exceptions and heavy fines for all caught with them (no exceptions), exotic reptiles could be effectively kept out of Australia indefinitely given Australia's huge law enforcement bureaucracy which rivals that of the former communist countries in Eastern Europe in terms of size.
The recent glut of exotic reptiles in Australia has been fuelled by several factors, including the fact that there is a general perception that they will soon be legalized (see the Weigel e-mails) and the fact that penalties for holding them illegally (if caught) are minimal.
In 2001, keepers Chris Hay and Rob Valentic were caught with a stash of illegal exotic reptiles, including highly venomous species, a hydroponic drug crop, stealing power from the supplier and illegal firearms.
The magistrate Alan Spillane gave them a good behavior bond (no effective fine, penalty or conviction). This was upheld in spite of an appeal to a higher court by the local wildlife officials. The precedent is currently the foundation for the continued illegal importation of exotic reptiles, in part fuelled by the larger zoos and the like who have shared in the proceeds, including from the Valentic/Hay bust.
Hence (for example) a case in June 2003 where another Victorian keeper charged with possession of illegal exotics was fined a few hundred dollars only, in spite of the reptiles he held being valued at many thousands of dollars.

NEW AND PREVIOUSLY UNKNOWN FINDINGS OF FACT IN RELATION TO OPMV

- In situ transmission is generally via fluids, not air. This means, blood, saliva, water bowls, bowl washers and mites. Most common means between cages is shared washing cloths for water bowls or transfer by mites. Less common is via saliva or fluids left on feeding tongs or forceps.
- OPMV effectively stops growth in young snakes for the duration of infection and is another hitherto unrecognized indicator of the virus.
- Feeding of otherwise non-feeding snakes by assist or force-feeding may be beneficial in terms of nursing ill snakes. OPMV affected snakes will generally hold down and digest food without problem at all stages of the infection except at the terminal (restless phase), even if they do not voluntarily take food.
- Snakes with neurological symptoms early on are likely to die, regardless of care, while those exhibiting only respiratory symptoms have a much greater chance of survival.
- Failure to properly thermoregulate is another factor indicative of OPMV.
- Progression of OPMV in snakes in laboratories AND in the captive situation is highly predictable based on identifying time of infection. Infection time can also be deduced by working backwards from when symptoms are detected if these parameters are known for the species.
- Within a given species, size (or lack thereof) directly indicates likelihood of surviving OPMV as does other factors in terms of general health and the presence or absence of other ailments.
- Contrary to inferences in some previously published reports, OPMV will affect neonate snakes. In fact this virus is far more deadly in small snakes than large.
- Contrary to previously published reports that snakes that are 'poor doers' are indicative of OPMV while non-poor doers are not, in terms of a single collection, the evidence here suggests that all snakes in a collection may have OPMV, but many will fail to show signs and be erroneously cleared as healthy when carrying OPMV. In other words, the poor do-ers with other pre-existing problems are much more likely to succumb to OPMV (as seen in the case of the two large *A. cummingi* received by Hoser on 15 Feb 2003).
- OPMV related deaths in snakes may be indicated by a snake's body being found in a loosely curled 'belly-up' position. For the species in the genera *Notechis* and *Acanthophis* at least, this is almost a general trend.
- OPMV infections in different collections can be easily and accurately predicted based on snakes known to be infected, movements between collections, an appraisal of cage cleaning, water sources, mites and feeding methods. This includes accurate identification of snakes carrying the virus but showing no symptoms.

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All reptiles mentioned in the paper above were held and/or moved under various permits as issued by the relevant state wildlife authorities.
Comments about husbandry practices of various keepers are made here in as frank and accurate a manner as possible and not to personally attack these people. For the purposes of this paper it has been essential to mention all relevant facts including such things as cage cleaning methods, mite infestations and so on.

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Trail of an OPMV infection

Notes: This is a partial listing only and it is only to end June 2003. The diagram illustrates the sequence of movements and events. More deaths are expected as many snakes in most collections remain seriously infected and/or were displaying symptoms.