

Snakebite in Papua New Guinea

Facts & Fiction

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Introduction

Snakes are widely feared in Papua New Guinea, and with very good reason. In many parts of PNG snakebite is an almost daily occurrence and venomous snakebite is a serious public health problem, with localized incidence rates that are among the highest of any tropical region in the world. Medical and epidemiological studies of snakebite in different parts of the country have given us detailed snapshots of some of the outcomes of snakebite, and although there are still many gaps in our knowledge, there is a significant amount of factual data available.

Ask anyone about snakebite and they will undoubtedly have a story to tell about someone they know who was bitten by a snake, or who died of snakebite. It would in fact be very easy to believe that venomous snakes lie in wait for unsuspecting people at every turn, and that right across PNG snakebite is claiming dozens of lives every day. If you ask Papua New Guineans about which snakes are responsible for snakebite one species above all others will feature at the forefront of every conversation; the fearsome ‘Papuan (Pap) black’.

Of course the challenge for clinicians, health workers and scientists when it comes to snakebite is to separate the finely woven threads of fact and fiction:

- *Just how many people really are bitten by snakes, and in what parts of the country do these bites occur?*
- *Are there as many deaths as either the scientific data, or the local people would have us believe?*
- *Which species are dangerous and bite the most people and which are not?*
- *What types of antivenom will best suit the needs of certain areas of the country?*

We have to be able to answer these and many other questions in order to provide the victims of snakebite with the most appropriate medical care. Having factual data enables resources (including antivenoms) to be distributed to the right places, and most importantly of all, reliable data provides health managers with the information they need to be able to make the right funding decisions to address community needs.

At the present time however the extent of our knowledge lacks national clarity; there are many provinces throughout the country for which we simply have no reliable data. Without even basic information it is extremely difficult to give clinicians and health workers exact information about snakebite in their communities, and it is completely impossible to provide sound advice to health administrators. As you will learn in this chapter, research into the epidemiology and clinical consequences of snakebite is taking place, and we have learned quite a lot about snakebite over the last fifty years. We hope to stimulate your enthusiasm to assist in ongoing research and to develop our knowledge even further.

Local beliefs & perceptions of snakes and snakebite

Snakes occupy an important role in local culture and tradition, and it is these beliefs and perceptions that affect the ways in which Papua New Guineans deal with the issue of snakebite and snakes in general. In many communities across PNG snakes are considered to be the mortal shells of bush spirits, demons, or deceased ancestors, enemies and sorcerers. There are numerous legends that tell of fearsome demons which take the natural form of snakes. The Marind-anim people from south-western PNG and eastern Papua believe, for example, that a demonic old woman known as the *pathogu* steals young children while in the body of a snake. To the Orokolo people living to the west of Kerema in Gulf province, snakes are the homes of mythical bush spirits – dark and dangerous creatures that can cause untold trouble.

Many Mekeo people believe that the dreaded ‘Papuan black’ attacks people at the behest of a sorcerer who may have first stolen something personal from the victim and have placed it into a heated pot containing the body of a snake. A similar belief is held by the Kiwai people from the Fly River delta; the *ove-devenar* sorcerer will collect the faeces of his victim and put it into the mouth of a snake model made from wood or clay which he then places in an area that the victim is likely to visit. The model becomes a living snake which hunts and kills the *ove-devenar*’s target. The Elema people of Gulf province believe in an evil spirit called *ove-hahu* who is the cause of accidents, serious sudden illnesses and snakebite. Similar traditional beliefs about snakes are held by the Arapesh people from the Sepik region, and by many other clans throughout PNG.

In Central province, the coastal Motu-Koitabu and mountain Koiari peoples often believed that snakebite, as well as being a tool of the *vada* (sorcerer), was sometimes a punishment meted out by good spirits such as the *birava* for social and traditional crimes like adultery. The Keakalo people of Marshall Lagoon also believed that snakebite was a common punishment for social transgressions such as adultery, theft, spousal abuse or taboo-breaking. As well as this, their *mega mega auri* (snake senders) could not only use magic to send out a snake to bite someone, but could also be sought out and paid to administer a cure. Dr Charles Campbell, a physician who conducted the first medical studies of snakebite in PNG learned that this cure usually involved obtaining a variety of tree bark called *paia* and a rainforest vine *wamela* which were chewed by the *mega mega auri* and then blown onto the face and body of the victim, who would also be massaged with coconut flesh or milk and *kiki* leaves. Chewing the *paia iwoa* (ginger leaves) and *wamela* was also considered to be a way of deterring snakes from biting.

Just as snakes are associated with evil deeds, dark spirits and sudden death, they are also widely held to play an important role in fertility and the guardianship of important traditional sites. The Kamea people (known as the Kukukuku by outsiders) live in the remote highlands of Gulf and Morobe provinces. These small, strongly built people, with an infamous warrior tradition and a fearsome reputation, have many animistic beliefs and some of them believe strongly that an enormous snake keeps the world functioning and protects us all. This creature is guarded by benevolent sorcerers who engage in a constant struggle to protect the snake from evil sorcerers who would kill it, a calamity that would spell the end of the world.

Kiwai people in Western province believe that the mythical *maigidubu* snake spirit is crucial to the success of yam crops, and that if the *maigidubu* leaves its tracks through the crop around the time of the yam festival, then success will be assured. Also, their guardian spirits the *etengena*, would often take the form of snakes and stand guard over their gardens, biting intruders to send them away.

Trobriand Islanders believe that some snakes are sacred and either house the spirits of great chiefs, or are their reincarnations. All the same, finding such as snake in a village was not considered a good sign, and unless the creature could be tempted to leave with prayers or other offerings, sudden illness was likely to befall the entire community. Despite this dreadful possibility, it was taboo to kill the snake, as this would only make the consequences worse.

The Bariai of West New Britain tell the tale of Moro, a snake-man who started the tradition of pig exchanges and other ceremonies in honour of firstborn children, and to give praise to the dead. Legend has it that Moro's father Kamaia had been killed in a disagreement with his brothers-in-law, and his liver cut out and cooked. Later during his funeral Moro was tricked into eating some of the liver by his cousin-in-law Kaukave whereupon his two legs immediately became fused together and the entire lower half of his body was transformed into that of a snake. Moro's transformation is ascribed to the vengeful punishment of his father's ghost for the having consumed the liver, albeit unknowingly. Pursued by the ghost of his father, Moro and his mother escape after Moro uses magic to carve out the Amara River, and then tricks the ghost into trying to cross, only to be eaten by a large crocodile!

Distinctions between different types of venomous snakes are made by many of the people living in southern PNG although different names may sometimes be used by different clans of the same language groups for exactly the same snake, and other groups use just one name to describe different venomous snakes:

Papuan taipan (*Oxyuranus scutellatus canni*)

Moveave (Gulf)	<i>Lavai</i> – 'black snake with a red stripe that lives in long grasses'
Mekeo (Central)	<i>Auguma</i> – 'black snake that bites again'
Motu-Koitabu (Central)	<i>Kabagi</i> – 'red snake' by the people of Barune <i>Larana Karo</i> – 'long blacksnake' by the Barune people <i>Duba</i> – dark coloured snake by the Kila Kila people
Keakalo (Central)	<i>Relena Gamara</i> – 'big brown snake'

Papuan blacksnake (*Pseudechis papuanus*)

Moveave (Gulf)	<i>Mito</i> – 'black snake that lives in Sago swamps'
Mekeo (Central)	<i>Auguma</i> – 'black snake that bites again'
Motu-Koitabu (Central)	<i>Larana Karo</i> – 'long blacksnake' by the Barune people <i>Duba</i> – dark coloured snake by the Kila Kila people
Keakalo (Central)	<i>Gelema rupa</i> – 'black snake'

Death adder (*Acanthophis* spp.)

Mekeo (Central)	<i>Afi</i> – 'sharp-eyed snake'
Motu-Koitabu (Central)	<i>Asenamo Api</i> or just <i>Api</i> – 'short sharp-tailed snake'
Keakalo (Central)	<i>Vanaame</i> – 'short snakes that jumps'

Neither the Mekeo nor Kila Kila Motu people make a significant distinction between taipans and blacksnakes and use just one name each to describe the two different species. Even the people of the Moveave region in the east of Gulf province, while using different names to describe taipans and blacksnakes, identify both as 'blacksnakes'. The majority of people are also able to distinguish venomous snakes from non-venomous pythons and other snakes. The Motu people know pythons as *Navara*, while their Koitabu cousins use *Lavara*, and the Keakalo call large pythons *Kapari* and may consider some of them sacred spirit animals.

When is a *black* snake not a blacksnake?

The use of colour to describe and identify snakes is common all over the world. In Australia snakes with stripes are called ‘tiger snakes’ (although the reality is that not all of them have stripes), and even in Papua the small-eyed snake (*Micropechis ikaheka*) is called a ‘tiger snake’ because of the striped appearance of some specimens. Brown-coloured snakes in Australia were given the obvious name ‘brown snake’ while black-coloured snakes are called ‘blacksnakes’; even though at least one member of the ‘blacksnake’ taxonomic group is called a ‘king brown snake’ (*Pseudechis australis*) because it is actually a brown-coloured ‘blacksnake’!

In Papua New Guinea people tend to do exactly the same thing. All black-coloured snakes are called ‘blacksnakes’ based entirely on the colour of their bodies. Whether or not they happen to really be venomous Papuan blacksnakes (*Pseudechis papuanus*) is an entirely different issue. There are in fact many different types of black-coloured snakes in Papua New Guinea, some of them non-venomous, some moderately venomous and three that are highly venomous and very dangerous; they include:

Black whipsnakes (<i>Demansia vestigiata</i>)	Moderately venomous
Boelen’s pythons (<i>Morelia boeleni</i>)	Non-venomous
Brown-headed snakes (<i>Furina tristis</i>)	Moderately venomous
Common tree snakes (<i>Dendrelaphis punctulatus</i>)	Non-venomous
D’Albert’s pythons (<i>Leiopython albertisi</i>)	Non-venomous
Death adders (<i>Acanthophis</i> spp.)	Highly venomous & dangerous
Forest snakes (<i>Toxicocalamus</i> spp.)	Moderately venomous
Javan file snakes (<i>Acrochordus granulatus</i>)	Non-venomous
Mangrove snakes (<i>Myron richardsoni</i>)	Moderately venomous
Muller’s crowned snakes (<i>Aspidomorphus Muelleri</i>)	Moderately venomous
New Guinea ground boas (<i>Candoia aspera</i>)	Non-venomous
Papuan blacksnakes (<i>Pseudechis papuanus</i>)	Highly venomous & dangerous
Papuan taipans (<i>Oxyuranus scutellatus canni</i>)	Highly venomous & dangerous
Slatey-grey snakes (<i>Stegonotus cucullatus</i>)	Non-venomous
Solomon’s coral snakes (<i>Salomonelaps par</i>)	Moderately venomous
Water pythons (<i>Liasis mackloti</i>)	Non-venomous

With so many different ‘Papuan (Pap) blacks’ distributed throughout the country it should be very obvious why this almost supernatural snake ends up being blamed for virtually every snakebite that occurs in PNG!

It should also be very clear why some bites by ‘Papuan blacks’ cause absolutely no clinical illnesses at all, while other may produce minor signs, and still others are lethal.

There is **ONE** snake in this list that really is a Papuan blacksnake (*Pseudechis papuanus*) both in colour and scientific identity. It is a highly venomous species, but the reality is that, of the venomous snakes in this list of sixteen different types of snakes, only three are highly venomous, and of these, the one which causes the majority of snakebites is not a ‘Papuan black’ at all, but the actually the much more dangerous and much more venomous Papuan taipan (*Oxyuranus scutellatus canni*). In Central province taipans cause more than 80% of all snakebites, while Papuan blacksnakes (*Pseudechis papuanus*) cause less than 5% of the serious snakebites admitted to PMGH.

Can you pick the REAL Papuan blacksnake?



A



B



C



D



E



F



G



H

Turn to the last page of this Chapter to see if your attempt at identification was correct or not.

Common misconceptions about snakebite

There are several common misunderstandings and misconceptions about snakes and snakebite in Papua New Guinea:

Belief: **The majority of snakebites are caused by the Papuan blacksnake.**

Reality: From a medical perspective, this is a particularly dangerous misconception that can seriously harm a snakebite patient.

The clinical reality is that there is little evidence to show that the **real** Papuan blacksnake (*Pseudechis papuanus*) causes large numbers of snakebites, particularly in Central province where investigations using venom identification assays have shown that only 4.3% of the serious snakebites admitted to Port Moresby General Hospital were caused by this species.

In Milne Bay, Gulf and Western provinces, it is still possible that a larger proportion of snakebites are caused by the Papuan blacksnake, but at the present there is very little reliable evidence available. Papuan blacksnakes do not occur in any other parts of Papua New Guinea.

In Central province the evidence suggests very strongly that the majority (more than 80%) of serious snakebites admitted to PMGH are caused by Papuan taipans (*Oxyuranus scutellatus canni*). In the past people have died as a result of the mis-identification of taipans as ‘blacksnakes’.

Belief: **The death adder uses a poison spine on its tail to harm people.**

Reality: This is completely untrue. The soft spine-like projection on the end of a death adders (*Acanthophis* spp.) tail contains nothing toxic, and has no role in the injection of venom.

The ‘spine’ is nothing more than a lure that is wriggled by the snake in order to attract food – just like dangling a worm on a fishing hook. When a small lizard or frog tries to eat the lure, the snake bites the animal killing it with venom injected through fangs in the roof of the mouth.

Belief: **The forked tongue of a snake is a poisonous sting.**

Reality: The tongue of a snake is completely harmless. It is a specialised scent organ that is used to collect odours from the air and deposit them in a special organ on the roof of the mouth (Jacobson’s organ) that contains the same types of olfactory cells that occur in the human nose, and which allow us to smell – the tongue does nothing more than help the snake detect smells and odours.

Belief: **All snakes are venomous.**

Reality: This belief could not be further from the truth; of the 112 species of snakes that occur in Papua New Guinea and Papua, only a third are venomous, and of 37 species, there are currently only 6 land snake species known to have the ability to cause human fatality. The two species of sea krait (*Laticauda* spp.) can also cause death, as can several species of true sea snakes; but sea snake related deaths are very rare in PNG.

Epidemiology of snakebite in PNG

Although the first published medical report of snakebite in Papua New Guinea did not appear in the literature until 1961, the risk to public health presented by venomous snakes was well known, and snakes generally were (and remain) widely feared.

While the Papua New Guinean perception of snakebite may have revolved around the traditional beliefs of the various cultural and social groups throughout the country, the perceptions of colonial medical officers was tempered by ‘western’ attitudes which had by the start of the 20th Century largely turned away from belief in supernatural forces towards acceptance of ‘scientific’ conclusions that were based on rational hypotheses, demonstrable facts and clinical reality. Significant advances in human understanding of basic physiology and biology provided a vastly different perspective of the causes of the clinical effects seen after snakebite. Rather than being considered as the result of sorcery, colonial doctors had a strong belief that snakebite was the consequence of the physiological changes produced by organic toxins.

Over the last 5 decades there has been considerable interest in the problems associated with snakebite in PNG, and a number of epidemiological and clinical studies, aimed at learning more about the consequences of snakebite and the outcomes of various treatment strategies, have been carried out. As a result there exists a considerable body of information to help us in understanding why snakebite occurs, what the consequences may be, and what we should be doing to improve the prognosis for snakebite patients.

Incidence and Mortality Rates

Early publications about snakebite in Papua New Guinea give no data on the overall incidence of morbidity or mortality, and concentrate predominantly on the reporting of case series that describe clinical syndromes of envenoming and their treatment. Campbell & Young (1961) reported 15 cases of serious envenoming from Central province between November 1959 and August 1960. The single death in this series was due to pulmonary oedema. Campbell (1964) reported an additional 41 cases in addition to further discussion of 11 cases from the earlier study. All of these cases occurred between mid-February 1960 and September 1962, with 38 cases originating within 80 kilometres of Port Moresby. As with the earlier paper, no data is presented to explain whether these represent all of the cases treated, or whether they only represent cases treated by the author.

Campbell also published three papers describing clinical syndromes of envenoming by presumed death adders *Acanthophis antarcticus*¹ (1966), Papuan taipans *Oxyuranus scutellatus canni* (1967a), and Papuan blacksnakes *Pseudechis papuanus* (1967c), but again gives no indications as to whether the cases reported represent all cases during particular periods, or are simply representative case histories. None of the cases were fatal. In a report on antivenom use Campbell (1967b) records a case fatality rate of 7.1% from 28 cases treated over a 21 month period from March 1964 to November 1965. In discussing the management of 73 cases between 1959 and 1965, Campbell (1969a) records 5 fatalities (CFR of 6.8%). Campbell (1969b) recorded mean admissions for snakebite in Papuan hospitals as 155.5 cases per annum for the period 1961-1967, and says that there were 6.3 admissions for snakebite per 1,000 patients (0.63%).

¹ *Acanthophis antarcticus* is not currently believed to occur in Papua New Guinea, and ongoing taxonomic investigations suggest that the genus is represented by several distinct species including *Acanthophis rugosus* and *Acanthophis laevis*.

Hudson & Pomat (1988) provide data on the incidence of snakebite in Madang province during the 10-year period from 1977 to 1986, but do not calculate actual incidence rates despite giving general population data. Using the data, the mean annual incidence can be calculated as 8.3 cases per 100,000 population. Clinical data for all of the cases is not available, and consequently any estimate of envenomation incidence would most likely underestimate the true figure. Only 2 deaths were recorded, giving a mortality rate estimate of 0.09 cases per 100,000 population and a case fatality rate of 1.14%. Brian & Vince (1988) examined snakebite in children admitted to Port Moresby General Hospital and reported a case fatality rate of 7.7% among 54 2-16 year olds over 38 months.

Currie *et al* (1991) produced incidence rates for envenoming snakebite and snakebite mortality in Papua New Guinea based on antivenom usage data from hospital admissions in Port Moresby (1987-1989) and Madang (1978-1988), and from CSL antivenom usage reporting forms (1983-1988). The authors acknowledged that these sources represented incomplete data on antivenom usage in Papua New Guinea. The annual incidence rates (January 1987-June 1989) of envenomation in rural central Papua and urban Central Papua were 81.8 and 21.8 cases per 100,000 population respectively. Corresponding annual mortality rates were given as 4.3 and 2.1 cases per 100,000 population. For the Madang region (1978-1988) they reported the annual envenomation incidence to be 3.0 cases per 100,000 population, with an annual mortality rate of <1.0 case per 100,000 population. Examined critically, these rates must underestimate the true rates, since antivenom supplies rarely meet demand, and many patients with envenomation never receive antivenom. Currie *et al* (1991) argue that the intention was to reduce diagnostic error by only considering cases in which antivenom was administered. A more appropriate design would have been a prospective study using enzyme immunosorbent assay technology to positively confirm the presence of venom antigen in the serum of patients in which snakebite was a suspected diagnosis.

Laloo *et al* (1995b) examined the epidemiology of snakebite in the Central province of Papua New Guinea as part of an extensive study supported by Oxford University and the Wellcome Trust. Retrospective analysis of admissions for snakebite at five rural health centres and at Port Moresby General Hospital (1987-1991) was combined with prospective Port Moresby General Hospital data for the period 1990-1992. Annual incidence and mortality rates for each of five sub-provinces were calculated as well as overall provincial rates. The annual incidence of snakebite ranged from 20.6 to 526.2 cases per 100,000 population at sub-province level. Provincial annual incidence was calculated as 215.5 cases per 100,000 population. The rate of actual envenoming was given as 62.6 cases per 100,000 population per annum, but no criteria used to identify these patients are provided. They reported that case fatality rates ranged from 2.9% to 30% at five rural health centres in Central province. For Central province annual mortality is reported as 7.9 cases per 100,000 population, and the combined Central province/National Capital District annual mortality is given as 3.7 cases per 100,000 population. From 1987 to mid-1992 the case fatality rate at Port Moresby General Hospital was 4.4% overall, however the rate in children under 10 years (10.0%) was significantly higher than the rate for adults (3.3%).

Williams *et al* (2003) studied snakebite admissions to health centres in the Mekeo region of Central province between 1997 and 2001 and reported that the average annual incidence of snakebite was 561.9 cases per 100,000 population. This figure compares very favourably to the rate of 526.2 cases per 100,000 population provided by Laloo *et al* (1995b). Localised community incidence rates reported by Williams *et al* (2003) were however significantly higher with three large communities having average annual incidence rates of between 1,041 and 1,448 cases per 100,000 population.

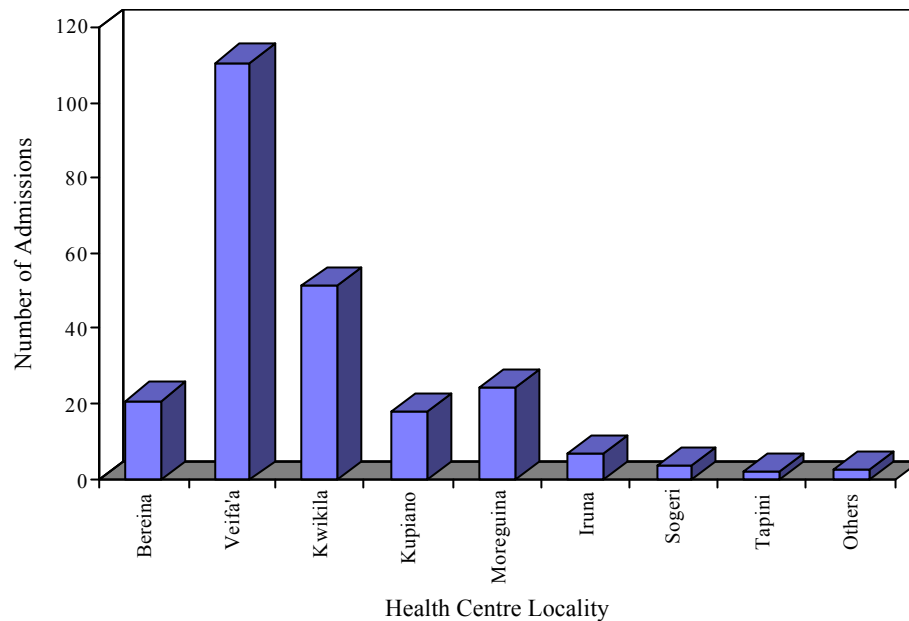


FIGURE 1: Average annual admissions for snakebite at major health centres in Central province 1987-1991 (SOURCE: Laloo *et al* 1995)

Not all snakebites resulted in envenomation. The average annual incidence rate for envenomation was 326.9 cases per 100,000 population; effectively 58.2% of snakebites resulted in the development of clinical envenoming. Annual mortality rates ranged from 7.5 to 19.3 cases per 100,000 population with an average mortality rate of 13.8 cases per 100,000 population. This is significantly higher than has been demonstrated in the past.

Williams *et al* (2002) determined case fatality rates for 283 patients admitted to the Intensive Care Unit at Port Moresby General Hospital for the treatment of snakebite between January 1998 and December 2001. The overall case fatality rate was 9.54% but annual case fatality rates ranged from 4.4% to as high as 20.6%. Case fatality rates among males (7.7%) were significantly different ($\chi^2=24.6$, $P<0.001$) to females (13.5%). There was also a significant difference ($\chi^2=37.4$, $P<0.001$) between case fatality rates of children² (15.7% overall; 14.3% for males; 17.9% for females) and older patients (7.5% overall; 5.9% for males; 11.5% for females). McGain *et al* (2004), examining snakebite mortality at Port Moresby General Hospital in the period from January 1992 to December 2001, reported a total of 87 deaths among 722 admissions for envenoming, giving a case fatality rate of 12.0%. The snakebite case fatality rate for ventilated children in the Intensive Care Unit was 16.2% compared to 10.2% for ventilated adults. At the present time no data on the incidence of morbidity or mortality following snakebite in other regions of Papua New Guinea is available.

Age Distributions and Sex Ratios

Campbell & Young (1961) and Campbell (1964, 1966, 1967a, 1967b) give an elementary profile of the epidemiology of snakebite during the 1960's. The mean age of 57 male victims was 26.8 years (SD=10.6 yrs) and 20 years (SD=9.8 yrs) for 10 female patients. Twelve patients (17.9%) were under 15 years of age. Patients ranged from 3 years to 45 years of age.

² Paediatric cases 10 years of age or under.

Hudson & Pomat (1988) examined 129 cases (83 males; 46 females) from Madang province for which medical records were available. There were 111 adults and 18 children under 12 years of age; however no further data on age distribution is given.

Currie *et al* (1991) report that the mean age of 347 patients (215 males; 132 females) in their study was 24.5 years (SD=13.8 yrs), but give no sex-related data. The youngest patient was 2 years old and the oldest was 67 years of age. Ninety patients (25.9%) were under 15 years of age, and 69% of these were males. The overall male:female sex ratio was 1.6:1. Lalloo *et al* (1995) state that the mean age of patients in their study was 25.0 years; but, like Currie *et al* (1991), give no sex-related data. Of the admitted patients, 8.5% were children 10 years old or younger, and children comprised 16% of envenomed and 6.9% of non-envenomed patients. Lalloo *et al* (1995b) report an overall male:female sex ratio of 1.39:1 based on 1,421 admissions to Central province health centres and Port Moresby General Hospital between 1987 and 1991. At Veifa'a in the north-west of the province the male:female sex ratio was 0.87:1. The lack of sex-specific age data in both of these large recent studies is unfortunate. In Lalloo *et al* (1995b) the mean age of all admitted patients (25.0 years) differs from the mean (21.1 yrs, SD=17.7) and median (15.5 yrs) ages of 20 patients who died from snakebite in their study. Median ages of male (14 yrs, n=11) and female (16 yrs, n=9) fatalities are at variance with the mean age given for all admissions. However, it is not possible to compare this data further as no sex-specific calculations are provided.

Williams *et al* (2003) do give age-specific data for snakebite in Mekeo, with a mean age for males of 23.1 years and 22.7 years for females and a median age of 20 years for both sexes. There were a disproportionate number of 16-30 year old males and females bitten by snakes in Mekeo; 49.7% of all male snakebite victims and 53.6% of all female snakebite victims in Mekeo fell into this age range despite that fact that only 27.5% of males and 27.1% of females in Mekeo were aged between 16 and 30 years. Although 43.5% of the male population, and 41.1% of the female population were aged between 0-15 years old, snakebite victims of these ages were under-represented; 28.6% of males and 23.4% of females were 0-15 years old.

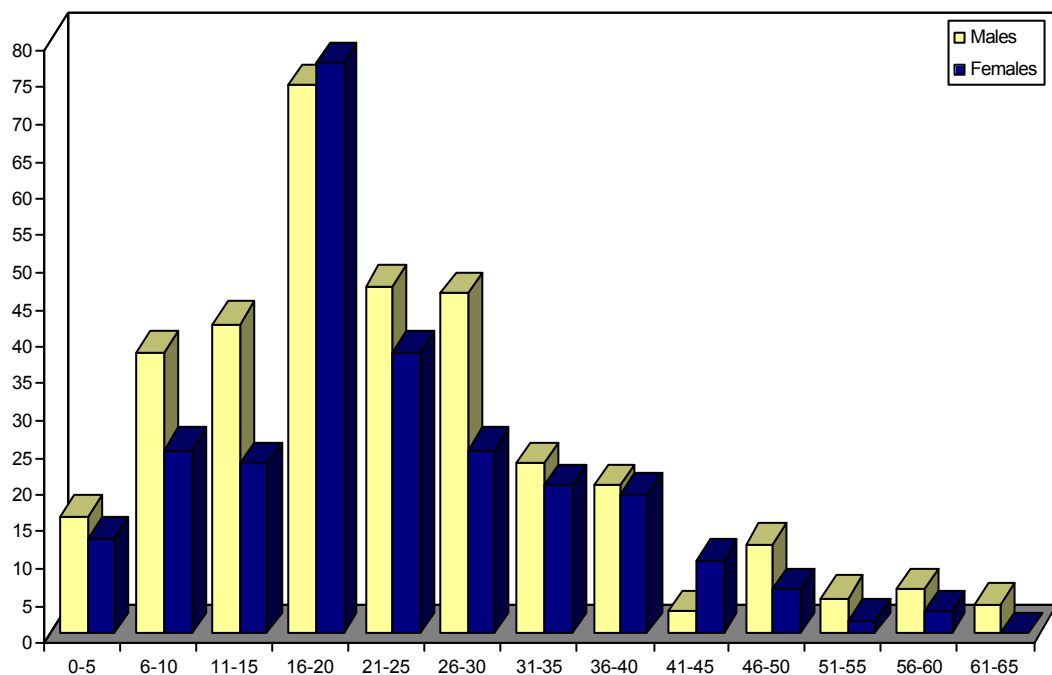


FIGURE 2 Age distribution of 336 male and 261 female patients admitted to health centres in Mekeo between 1997 and 2001 for whom actual age was recorded (SOURCE: Williams *et al*, 2003)

Williams *et al* (2002) give age and sex-specific data for their series of 283 seriously envenomed patients at Port Moresby General Hospital. Mean age for males was 21.5 years (n=170, SD=14.9 yrs, median=16 yrs) and 21.1 years (n=77, SD=16.5yrs, median=14 yrs) for females. Children 15-years old or under comprised 39.9% of cases. The overall male:female sex ratio was 2.2:1. However, in the 15-29 year age group the ratio of males to females was 4.1:1. Males under the age of 30 years comprised 46% of cases. McGain *et al* (2004) examining fatal snakebite at Port Moresby General Hospital report that 47% (n=41) were under 15 years of age, and that males were involved in 53% (n=46) of all cases.

Circumstances of Injury

In the first report on snakebite in Papua New Guinea, Campbell & Young (1961) state that all 15 patients were bitten on either the foot or the leg during daytime hours while the victims were either walking in or near long grass. Campbell (1964) reported that all but 2/52 patients were bitten in daylight hours; 96% of cases were from Central province and 73.1% came from within 80 kilometres of Port Moresby. All but one bite occurred on the lower limbs with 17.3% on the toes, 32.7% on the foot, 15.4% on the ankle and 32.7% on the legs themselves. Bites occurred when the victims were walking on paths near long grass, or through long grass, and 23% actually trod on the snake itself.

In his series of 15 cases of *Acanthophis* spp. bites over a six-year period, Campbell (1966) reported that all but one bite occurred on the lower limbs, typically on either the dorsum of the foot or the toes. Eight bites happened when the person stood on the snake. In one case a man laid down on a snake concealed beneath leaf litter and was bitten on the leg, and another was bitten on the finger by a snake he accidentally picked up in a pile of cut grass. The other 13 bites were on either the feet or ankles. Three cases involved patients bitten between 6.30pm and 7.30pm, while a fourth patient was bitten at 3.00am in the morning. The other cases occurred between 8.30am and 5.30pm. In the only other study of proven *Acanthophis* spp. envenomation in Papua New Guinea, Lalloo *et al* (1996) found that 22 of the 32 EIA-confirmed cases occurred in daylight, and that only two bites did not occur on the lower limbs.

Reporting bites by *Oxyuranus scutellatus canni*, Campbell (1967a) shows that all bites occurred on a lower limb (one of these was bitten on the “middle third” of the leg, and another on the “lower third”), and occurred between 10.00am and 4.00pm. Lalloo *et al* (1995a) observed that 85% of proven *Oxyuranus scutellatus canni* bites occurred during daylight among people working in gardens or walking along bush tracks, and that 96.4% of all the bites were to the lower limbs. All nine cases of presumed *Oxyuranus scutellatus canni* envenomation reported by Williams & Bal (2003) took place during daylight and involved bites to the legs, ankles or feet. One patient was bitten at home, two were bitten in the garden and the other six cases occurred when patients were walking along bush tracks or dirt roads.

Among Campbell’s (1967c) 13 cases of presumed *Pseudechis papuanus* bites, most (n=10) were bitten on either the foot or ankle, while 2 were bitten on the hand or arm and 1 was bitten on the shoulder. Positive identification of the biting snake was only made in one case (specimen brought to PMGH), and all of the other cases were more likely to have been caused by Papuan taipans (*Oxyuranus scutellatus canni*). Only two positive identifications of *P. papuanus* snakebite were made during Campbell’s 7 years at PMGH, and his contention that it was the most common cause of snakebite in southern PNG is almost certainly erroneous. All of the bites occurred in daylight. All nine cases of snakebite involving *Pseudechis papuanus* discussed in Lalloo *et al* (1994) involved bites to the lower limb that occurred during daylight.

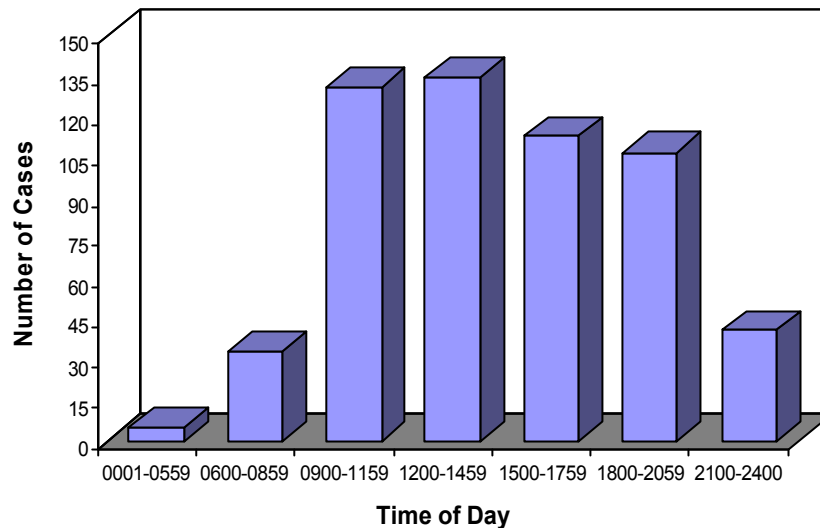


FIGURE 3 Time of day for snakebites in the Mekeo region of Central province between January 1997 and December 2001; increased frequency (43.9%) during the afternoon (1200-1759 hours) was statistically significant ($p < 0.005$) making this the major danger time for snakebite.

(SOURCE: Williams *et al*, 2003).

On the northern side of PNG, Hudson & Pomat (1988) reported that 81% of patients in their series of 129 patients were bitten on a lower limb; and that 87.6% of these were bites on the feet. Ten of the 129 patients (7.75%) were bitten on the hands. Sixty-two% of the bites occurred during the day and 33% in the night, while no time was recorded for 5% of the series. In Blasco & Hornabrook's (1972) report of a case of fatal *Micropechis ikaheka* envenomation, the patient was bitten at approximately 6.00pm while alone in the bush, and in Warrell *et al*'s (1996) case series of 11 bites by this species, which were confirmed by venom-antigen specific EIA, 3 of 7 cases for which a time of injury was recorded occurred after 8.00pm at night. One of the two patients who died was killed after being bitten while playing with the decapitated head of a snake he "killed" in the bush. Six of the patients in this series stood on the snake, and two were handling snakes they had found.

Currie *et al* (1991) found that 281 (81%) of 347 snakebite victims were bitten between 6.00 am and 6.00 pm, and that 53 bites (15.3%) occurred at night. Of 334 patients for whom data was available, 316 (94.7%) were bitten on the lower limbs; 204 of these (64.5%) were bites on the foot or the toes. No data was provided regarding what the victims were doing when bitten. Lalloo *et al* (1995b) report details for 335 cases of snakebite. The majority (89.4%) of 205 envenomed patients were bitten during daylight compared to 54% of the 130 non-envenomed patients. As with other studies, 86% of all bites occurred on the lower limbs. The three most common circumstances under which bites occurred were while victims were walking (34.5%), in the garden (19%) or in the bush (14.8%). In 86 cases of fatal snakebite discussed in detail by McGain *et al* (2004), 89% of the bites occurred on the lower limbs, and 60% of the bites for which time of injury was recorded occurred between 12.00pm and 6.00pm.

Identification of Biting Species

Throughout Papua New Guinea there are only four species of venomous snake that contribute to the majority of serious envenomations, along with perhaps two other species that can cause human fatality. The best information available to date shows that on the basis of

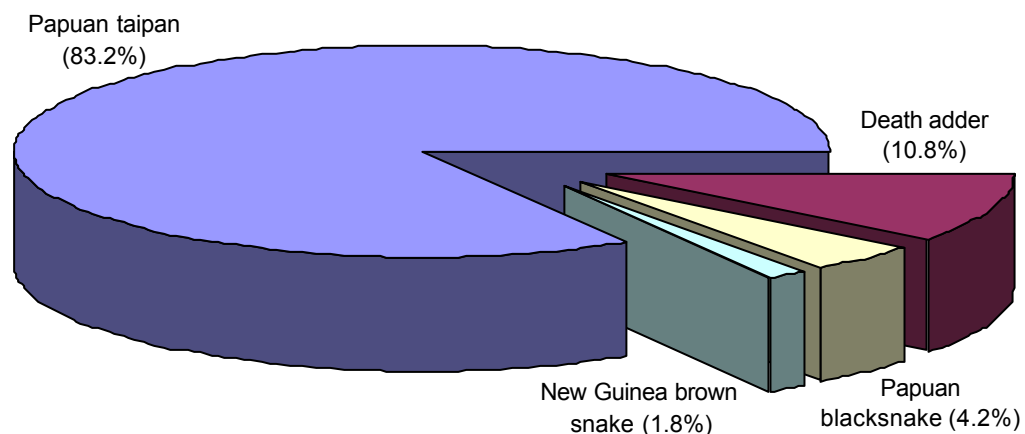
identifications made with the aid of species-specific venom enzyme immunosorbent assay (EIA) by Laloo *et al* (1995b), the snake responsible for the majority of serious snakebites admitted to PMGH is the Papuan taipan (*Oxyuranus scutellatus canni*) and not the Papuan blacksnake (*Pseudechis papuanus*). Papuan taipans caused an incredible 83.2% of envenomations compared to just 4.2% that were attributable to Papuan blacksnakes.

Reliable identification of the biting species is a major shortcoming in most studies of Papua New Guinean snakebite. In the 1950's herpetologist Ken Slater believed that the Papuan black snake *Pseudechis papuanus* was the commonest venomous snake in southern PNG, and even today, the perception of Papua New Guineans is that this snake is responsible for almost all snakebites. Slater (1956) wrote that it was most abundant along the coastline to the east of Port Moresby, and Campbell (1967c) claimed that it was responsible for more hospital admissions than any other species. In spite of this statement only two cases seen by Campbell between 1959 and 1967 could be conclusively attributed to this species on the basis of positive identification of the biting snake at the hospital. Campbell's (1967c) description of a syndrome of envenoming attributable to Papuan black snake *Pseudechis papuanus* bites must therefore be treated with scepticism.

The much more dangerous Papuan taipan *Oxyuranus scutellatus canni* often has a distinctive reddish-orange vertebral stripe as a distinctive feature, and Campbell appears to have relied upon this colouration as a means of presuming that large dark coloured snakes *apparently* lacking it were *Pseudechis papuanus*.

Although the Papuan taipan is the only large snake in PNG that may have a red or orange vertebral marking, this is a feature that many snakebite patients simply do not notice in the heat of the moment. There are no reliable means of visually distinguishing *all* specimens of *Pseudechis papuanus* and *Oxyuranus scutellatus canni*. One patient in Campbell's (1967c) series of 13 victims presented with a dead 1.5 metre snake for identification. Identification in the other 12 cases appears based on non-reporting of a vertebral stripe and these cannot be accepted as confirmed bites by *Pseudechis papuanus*. The only reliable recent description of envenomation by *Pseudechis papuanus* was given by Laloo *et al* (1994) who detailed the clinical syndromes of 9 patients bitten between January 1990 and June 1992 identified by specific venom-antigen enzyme immunosorbent assay.

FIGURE 4: Species responsible for serious snakebites admitted to Port Moresby General Hospital between January 1990 and June 1992 (SOURCE: Laloo *et al.* 1994, 1995)



Campbell (1964) states that 5 patients in a series of 52 arrived at hospital with a dead snake - 3 with death adders *Acanthophis* spp, 1 with a Papuan black snake *Pseudechis papuanus* and 1 with a Papuan taipan *Oxyuranus scutellatus canni*. Campbell (1966), describing the clinical syndrome of death adder *Acanthophis* spp envenoming reports 15 cases. The biting snake was killed in 11 of these cases and 8 of the victims brought the snake to the hospital where it was identified. Death adder envenoming is presumed in 5 cases on the basis of statements by either the patient or a third party, and in the last 2 cases Campbell presumes identification on the basis of haematological tests. Death adders have a distinctive body plan and appearance that makes visual identification easier than for other species. Lalloo *et al* (1995b) used species-specific venom EIA and found that 10.8% of serious snakebites admitted to PMGH were caused by death adders. Enzyme immunosorbent assay confirmed 32 cases of envenomation by *Acanthophis* spp. that involved a syndrome of progressive neurotoxicity without coagulopathy (Lalloo *et al*, 1996), and the 15 cases reported by Campbell (1966) conform to this profile, lending support to Campbell's diagnosis. Hudson & Pomat (1988) give an excellent case report of a death adder bite treated at Madang Hospital where the dead snake was identified at the hospital.

Campbell (1967a) reports six cases that he believed were attributable to envenoming by Papuan taipans *Oxyuranus scutellatus canni* based on positive identification of two dead snakes brought in with the victims, and a description of an orange or red coloured vertebral stripe in the remaining four cases. Lalloo *et al* (1995a) examine the syndrome of envenoming by *Oxyuranus scutellatus canni* in detail on the basis of 166 cases in which this species was implicated by specific venom-antigen EIA. Although they report the specificity for specific taipan venom-antigen as 87%, only cases in which this was the sole antigen detected were regarded as cases of definite *Oxyuranus scutellatus canni* snakebite. Williams & Bal (2003) reported nine cases of presumed envenoming by *Oxyuranus scutellatus canni* on the basis of clinical presentation and/or description of the biting species. In the future it is hoped that the widespread use of venom detection kits to diagnose snakebite will make it possible to assemble nationwide data on the species responsible for envenomation.

Hudson & Pomat (1988) reported on 64 cases of envenomation reported in Madang Province between 1977 and 1986; 15 presented with symptoms suggestive of bites by the small-eyed snake (*Micropechis ikaheka*). Warrell *et al* (1996) reported that in a more recent series, 4/46 (8.7%) cases of snakebite treated at Madang Hospital, and 5/13 (38.5%) treated at Gaubin Hospital on nearby Karkar Island were identified by EIA as attributable to *M. ikaheka*. No cases of *M. ikaheka* were identified in any of the patients studied using EIA by Lalloo *et al* (1994). Brown snake *Pseudonaja textilis* spp. envenomation has been reported among 1.8% of EIA-tested snakebite patients admitted to Port Moresby General Hospital between January 1990 and June 1992 (Lalloo *et al*, 1994).

Climate and Seasonality

There have been few attempts to determine whether bites by certain species are more likely to occur under specific climatic conditions (temperature, rainfall, etc.) or during particular seasons throughout the year.

Campbell (1966) reported that for *Acanthophis* spp., 12 of the 15 cases occurred between August and December. Campbell (1967a) reports *Oxyuranus scutellatus canni* bites in June (2), August (1), December (1), January (1) and March (1). Currie *et al* (1991) reported that 56.8% of snakebites occurred between the 'wet season' months of from November to April.

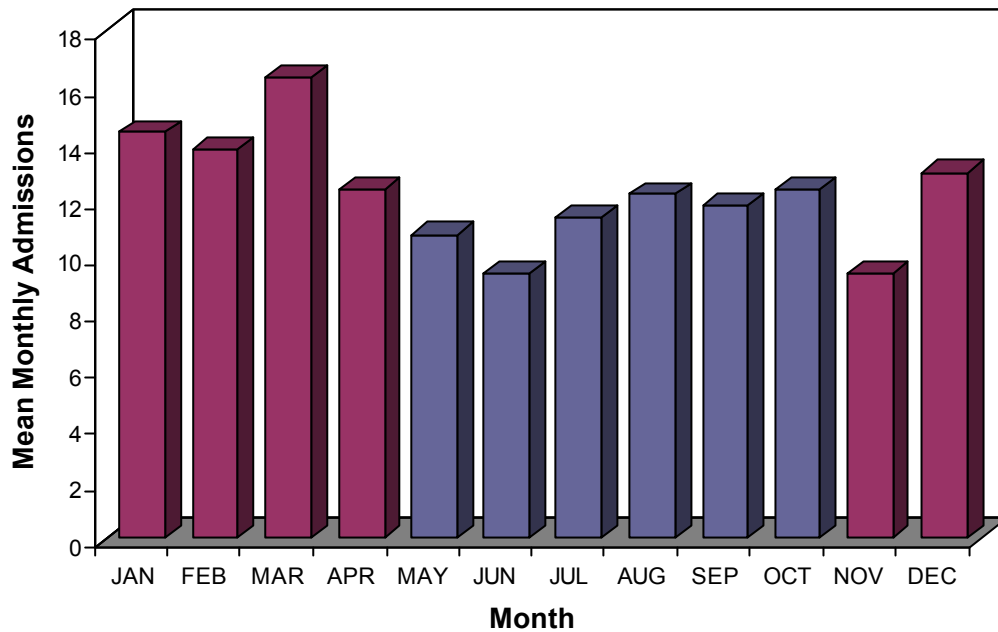


FIGURE 5: Seasonal frequency of admissions for snakebite in the Mekeo region of Central province between January 1997 and December 2001; increased frequency during the dry season months of November to April is statistically significant ($p=0.04$). (SOURCE: Williams *et al* 2003).

Lalloo *et al* (1995b) state that 57.9% of 221 envenomed patients and 51.6% of 128 non-envenomed patients were bitten between November-April. Data on monthly admissions for snakebite were compared to monthly rainfall; however rainfall itself was not shown to be a significant contributing factor.

Williams *et al* (2002), reporting cases admitted to the PMGH ICU from January 1997 to December 2001 from across PNG, found that 54% occurred between November-April, and Williams *et al* (2003) found that 53.9% of snakebites in Mekeo occurred during the same months during this same period. McGain *et al* (2004) reported that 60% of fatal cases in the 10 years from January 1992 to December 2001 occurred between November and April.

Clinical Epidemiology and Envenomation Syndromes

Local Indications of Envenomation

The clinical syndromes of envenomation after snakebite in Papua New Guinea have been reported by several authors. Most reports relate to small case series of patients and, in most, the identification of the biting species is equivocal. Some of the consistently reported symptoms of snakebite over the last 40 years are shown in Table 1.

Lymph node pain is a consistent feature after snakebite in Papua New Guinea, and tender enlargement of regional lymph nodes is reported in all published accounts. Venom injected subcutaneously typically enters the lymphatic system and drains via lymph nodes into the circulation, and tenderness or swelling of lymph nodes may be an indication of venom absorption. Trevett *et al* (1994a) stated that lymphadenitis was a particularly sensitive positive indication of envenomation and occurred in 93% of envenomed patients.

TABLE 1: Symptoms reported in various studies of snakebite in Papua New Guinea between 1964 and 2004.

Author	Local Pain	Lymph Nodes	Oedema	Headache	Vomiting or Nausea	Abdominal Pain	Spit/Vomit Blood	Visual Disturbances
Campbell & Young, 1964	+	+	+	+	+	+	+	+
Campbell, 1964	+	+	+	+	+	+	+	+
Campbell, 1966	+	+	+	+	+	-	-	+
Campbell, 1967a	-	+	-	+	+	+	-	-
Campbell, 1967c	+	+	+	+	+	+	+	+
Campbell, 1969a	-	+	-	+	+	+	+	-
Hudson & Pomat, 1988	+	+	+	+	+	+	-	+
Laloo <i>et al</i> , 1994	+	+	+	+	+	+	+	+
Laloo <i>et al</i> , 1995a	-	+	-	+	+	+	+	+
Laloo <i>et al</i> , 1996	-	+	-	+	+	+	-	+
Warrell <i>et al</i> , 1996	+	+	+	+	+	+	-	+
Williams & Bal, 2002	-	+	-	+	+	+	+	+
Williams <i>et al</i> 2003	+	+	+	+	+	+	+	+
McGain <i>et al</i> 2004	-	+	-	+	+	+	+	+

The extent and severity of local symptoms such as pain or oedema is significantly lower than in reports of elapid snakebite from Australia. Campbell & Young (1961) reported severe local pain and oedema in a single patient; however this was most likely caused by prolonged tourniquet use. Three other patients experienced minor oedema, and local pain was either absent completely or mild. Campbell (1964) found severe pain in 3 patients and severe oedema occurred in two individuals as a result of lengthy tourniquet application.

A third of the patients bitten by death adders *Acanthophis* spp. in Campbell (1966) had local pain, and 27% had slight oedema. Neither symptom was present in any of the 6 patients with *Oxyuranus scutellatus canni* envenomation reported by Campbell (1967a); however severe pain, oedema and extensive ecchymoses were seen in a patient bitten on the shoulder (Campbell, 1967c) and this may have been due to a Papuan blacksnake (*P. papuanus*) bite.

Pain was a feature in 36% of cases reported by Hudson & Pomat (1988) and 14% of patients had local oedema and localised muscle pain, most probably associated with rhabdomyolysis after *Micropechis ikaheka* envenomation. One patient in a series of eleven bitten by *Micropechis ikaheka* reported local pain and two patients had oedema (Warrell *et al*, 1996). Laloo *et al* (1994) observed slight oedema in one patient bitten by a Papuan black snake *Pseudechis papuanus*. In a series of 166 bites by *Oxyuranus scutellatus canni* (Laloo *et al*, 1995a) there were no reports of local pain or swelling. Clinical experience in Australia is that the bites by members of the blacksnake genus *Pseudechis* are painful locally and often associated with local oedema that may be mild to pronounced in severity. Williams *et al* (2004) reported severe local pain after a bite by an Australian Collett's blacksnake (*Pseudechis colletti*) and Isbister & Currie (2003) presents a case of mulga snake (*Pseudechis australis*) envenomation in a child with persistent bite site pain and swelling. Although uncommonly reported in PNG (perhaps because bites are uncommon) it should be anticipated that Papuan blacksnake (*Pseudechis papuanus*) and Papuan mulga snake (*Pseudechis cf.*

australis) bites may also produce significant local pain and oedema. Local pain and swelling caused by snakebite should not be confused with the pain and swelling that can be associated with ischemic injury due to prolonged tourniquet use.

Non-specific Indications of Envenomation

A number of non-specific features have been reported consistently in patients with snakebite envenomation. Campbell & Young (1961), and Campbell (1964; 1966; 1967a; 1967c; 1969a) reported that headache often followed envenomation, and headache has been universally reported in virtually all published accounts of snakebite in PNG. Headaches are reported to have last from a few hours to several days. Hudson & Pomat (1988), Lalloo *et al* (1996) and Warrell *et al* (1996) report headache as common following bites by death adders (*Acanthophis* spp.) and small-eyed snakes (*Micropechis ikaheka*). Herpetologist Ken Slater had persistent severe headache for two days after a bite from *Micropechis ikaheka*. Lalloo *et al* (1994) noted headache in 2 of 9 patients with confirmed Papuan blacksnake (*Pseudechis papuanus*) bite, and Lalloo *et al* (1995a) reported headache in 54.5% of positively identified Papuan taipan (*Oxyuranus scutellatus canni*) victims. In an analysis of referral letters written by rural health workers during the early 1990's, Trevett *et al* (1995) found that headache was reported in 37% of patients at the health centre, and in 58% of the same patients on admission to PMGH. Headache was reported in 51.9% of 564 patients bitten by snakes in Mekeo (Williams *et al* 2003). Headache was a documented non-specific symptom in 38 (63.3%) of 60 fatal snakebites treated at PMGH between 1992 and 2001 (McGain *et al*, 2004).

Vomiting and abdominal pain or tenderness typically occurs in unison. Campbell & Young (1961) observed concurrent vomiting and abdominal pain in 3 patients, while another 3 patients had vomiting without abdominal pain. One patient with severe abdominal pain and vomiting in conjunction with abdominal tenderness was mistakenly diagnosed with acute appendicitis. Vomiting was reported in 42% and abdominal pain in 34.6% of patients described in Campbell (1964). Vomiting was a typical non-specific indication in patients of death adder (*Acanthophis* spp.) bites reported by Campbell (1966), and was also recorded in three patients with presumed Papuan taipan (*Oxyuranus scutellatus canni*) bite (Campbell, 1967a). In the series of 13 bites dubiously ascribed to Papuan blacksnake (*Pseudechis papuanus*) envenomation, but most probably really having been caused by Papuan taipans (*Oxyuranus scutellatus canni*), 8 patients had early vomiting and 4 had severe abdominal pain (Campbell, 1967c). Vomiting after bites from the small-eyed snake (*Micropechis ikaheka*) has been reported by Blasco & Hornabrook (1972) and Warrell *et al* (1996). Abdominal pain was reported in 2 of the 6 patients reported in Warrell *et al* (1996). Hudson & Pomat (1988) reported abdominal pain in 56% and vomiting in 25% of 64 envenomed patients treated for either death adder (*Acanthophis* spp.) or small-eyed snake (*Micropechis ikaheka*) bites in Madang during the 1980's.

Of 9 patients with confirmed Papuan blacksnake (*Pseudechis papuanus*) envenomation, 2 vomited and 5 had abdominal pain (Lalloo *et al*, 1994). In a large series of Papuan taipan (*Oxyuranus scutellatus canni*) bite victims, 64.4% vomited and 59.8% had abdominal pain. In the analysis of referral letters conducted by Trevett *et al* (1994a), 38% had abdominal pain and 30% had vomited at the health centre; on admission to PMGH 58% had abdominal pain and 62% had reported vomiting. Williams & Bal (2003) reported vomiting in 4 of 9 patients with likely Papuan taipan (*Oxyuranus scutellatus canni*) envenomation and 3 of these 4 patients had concurrent abdominal pain. Williams *et al* (2003) reports abdominal pain in 48.9% of 564 patients bitten by snakes in Mekeo; however, nausea or vomiting was only reported for 6.2%. Abdominal pain was documented in 48 (80%), and vomiting in 40 (66%) of the 60 fatal snakebites discussed by McGain *et al* (2004).

Specific Indications of Envenomation: Coagulation defects

Evidence of coagulopathy is an absolute indication for antivenom if observed in conjunction with a history of snakebite in PNG.

Campbell & Young (1961) reported that 6 of the 15 patients had 'bleeding tendencies' and that 3 patients vomited blood-stained material. Campbell (1964) notes that 11/52 (21.2%) patients were recorded as either spitting blood or having haematemesis, while 3 patients also passed 'blood-stained' urine. Campbell (1967a) notes bleeding problems in 2 of 6 patients; one of these had persistent bleeding from a superficial facial wound, while another had haemoglobinuria and incoagulable blood. Haematemesis was seen in 3 patients and another 2 had haemoptysis among the group of 13 that Campbell (1967c) thought to have been bitten by Papuan blacksnakes (*Pseudechis papuanus*). Three of 4 patients in this series had haemoglobinuria and fibrinogenolysis, and other signs of coagulopathy (spitting blood; gingival bleeding; bleeding bite wounds) were reported in 8 of the 13 patients. As has been stated before, Campbell's presumptive identifications were based on whether or not the patient reported seeing a red marking on the back of the snake, and while the presence of reddish-orange marking is a strong indication that the culprit was a Papuan taipan (*Oxyuranus scutellatus canni*), it is important to again emphasize that not seeing this type of marking does not mean the snake was not a Papuan taipan.

Spontaneous bleeding has been reported in two cases of envenomation by the small-eyed snake (*Micropechis ikaheka*) that was reported in Warrell *et al* (1996). Hudson & Pomat (1988) did not report bleeding disorders in any of the 64 cases of snakebite from Madang province during the 1980's. In a study of 32 patients shown to have been bitten by death adders (*Acanthopphis* spp.) by EIA, there was no clinical evidence of coagulopathy although elevation in leucocyte counts and slight thrombocytopenia was found on laboratory investigation (Lalloo *et al*, 1996). Prothrombin time was significantly prolonged in 13 patients. Broad clinical experience in Australia and PNG indicates that bites by these snakes do not involve bleeding disorders.

Blood-stained saliva and bleeding from the mouth or nose was reported in 1 patient with confirmed Papuan blacksnake (*Pseudechis papuanus*) envenomation, while 2 others had either epistaxis or gingival bleeding (Lalloo *et al*, 1994). Laboratory studies on blood from 2 of these patients showed a mild thrombocytopenia (platelet counts: $91-93 \times 10^9/L$), and that three patients had lower than normal levels of several important clotting factors including protein C, fibrinogen, Factors V, VIII, XIII_a, anti-thrombin III, plasminogen and α_2 -antiplasmin. Prothrombin time (PT) and activated partial thromboplastin times (APTT) were prolonged briefly and FDP was elevated.

Coagulopathy can be a very prominent feature of bites by Papuan taipans (*Oxyuranus scutellatus canni*), and in addition to Campbell's early reports of bleeding after bites by large dark coloured snakes, there is a substantial body of evidence from recent studies. Lalloo *et al* (1995a) report that 77% of patients with EIA proven bites by Papuan taipans had incoagulable blood on the basis of 20WBCT > 20 minutes. Systemic bleeding occurred in 43.7% of patients, and bite site bleeding occurred in 16.3%. Most reported bleeding from either the gingival sulci (37%) or by haematemesis (12.6%). Three patients had epistaxis; 11% bled from superficial cuts and scratches; 8.8% bled from venepuncture sites. On laboratory investigation 52.8% had moderate to severe leucocytosis and 27.5% developed mild thrombocytopenia.

Brian & Vince (1987) reported that 40 of 54 children bitten by snakes developed evidence of coagulopathy and that 33 had clotting times greater than 15 minutes. Bleeding problems were

mentioned in 40% of referral letters written by rural health workers, and 60% of the referred patients were found to have incoagulable blood upon admission to PMGH (Trevett *et al*, 1994a). According to Trevett *et al* (1994a), a 20WBCT > 20 minutes had a positive predictive value (PPV) of 93.5% for a diagnosis of Papuan taipan (*Oxyuranus scutellatus canni*) envenomation. Williams *et al* (2003) reviewed 553 cases of snakebite from the Mekeo region in which the 20WBCT was used, and found that this simple diagnostic test had a positive predictive value of 98.4% and a specificity of 99.6% for the subsequent development of neurotoxicity. The PPV for subsequent coagulopathy was 67.7% with specificity of 95.7%; however, because the other signs and symptoms of incoagulable blood were often not reported in the clinical notes, it is probable that the actual PPV for disseminated intravascular coagulopathy is actually higher. In 60 fatal cases of snakebite reported by McGain *et al* (2004), 75% had a 20WBCT > 20 minutes and 35 patients (53.3%) had demonstrable spontaneous bleeding.

Specific Indications of Envenomation: Myotoxicity

There are few early reports of myotoxicity following snakebite in PNG, although some cases with 'haemoglobinuria' may actually have been patients with myoglobinuria instead. Blasco & Hornabrook (1972) mention that Ken Slater had 'weakness' after a bite from the small-eyed snake (*Micropechis ikaheka*), and this is often a sign of rhabdomyolysis. Hudson & Pomat (1988) report renal failure in 6 patients from Madang province who presented with dark-coloured urine. Hudson (1988b) reported two cases of presumed small-eyed snake (*Micropechis ikaheka*) envenomation. A 13 year-old girl bitten on the finger developed generalized muscle pain and tenderness and had urine that was dark reddish-brown, but was erythrocyte-free; and a 50 year-old man bitten on the finger while reaching into a bandicoot burrow by a 'black-headed snake with a white belly' also developed muscle pain and passed dark urine. This second patient's muscle pain was mild initially but became more severe over the next 2 days; at Modilon hospital in Madang his urine was grossly discoloured, but once again was erythrocyte-free. He received one ampoule of death adder antivenom and three ampoules of polyvalent antivenom. Both patients were said to be anicteric and without coagulopathy, and had neurotoxicity; the 13 year old girl subsequently died. Other cases of presumed *Micropechis ikaheka* envenoming treated in Madang also showed signs and symptoms of generalized rhabdomyolysis and myoglobinuria. Warrell *et al* (1996) describes classical signs of myolysis (generalised muscle pain and tenderness; trismus; dark urine) in three patients with EIA proven small-eyed snake envenomation.

The creatine kinase (CK) levels of 8 out of 12 patients bitten by death adders (*Acanthophis* spp.) was elevated (164-4,220 IU/L). Similar studies in victims of Papuan taipan (*Oxyuranus scutellatus canni*) bites found that creatine kinase levels were raised in 74.7% (43-8,110 IU/L) and levels of the liver enzyme aspartate transaminase (AST) were >50 IU/L in 52.6% of patients. Serum myoglobin exceeded 80 ng/ml in 52.2% of patients at the time of their admission. The serum creatine kinase levels of 2 of the 9 patients with proven Papuan blacksnake (*Pseudechis papuanus*) envenomation were slightly elevated; however there were no indications of rhabdomyolysis in any patient (Lalloo *et al*, 1994). In Australia, gross rhabdomyolysis and myoglobinuria after bites by members of the *Pseudechis* genus are relatively common, and creatine kinase levels may exceed 200,000 IU/L. Acute renal failure is a potential complication of myotoxicity, and McGain *et al* (2004) reported muscle pain in 41.7% of 60 fatal snakebites treated at PMGH. Renal complications were contributing factors in the deaths of 16 (26.7%) patients. Testing for creatine kinase, lactate dehydrogenase and other isoenzymes that quantitatively measure rhabdomyolysis products is not routine in PNG at present so it is possible that this important syndrome is seriously under-reported.

Specific Indications of Envenomation: Neurotoxicity

The neurotoxic effects of Papua New Guinean snake venoms are well defined in the available literature, and the balance of evidence demonstrates that snake venom neurotoxins are the major contributors in the deaths of patients due to the development of progressive flaccid paralysis that inhibits and compromises respiration.

The earliest signs of neurotoxicity often manifest themselves in the craniofacial nerves producing a sequential syndrome that often starts with diplopia and ptosis before progressing to ophthalmoplegia, dysarthria, dysphagia and bulbar palsy and eventually involving the intercostal, diaphragm and peripheral musculature. Ataxia and progressive muscle weakness results in a loss of locomotor function; patients eventually lose the ability to walk, stand, sit or even raise their limbs, or control head movements ('broken neck syndrome'). Deep tendon reflexes may be lost and failing respiration often leads to 'abdominal' breathing.

Campbell & Young (1961) graded the neurotoxic effects in 15 snakebite victims according to severity. Ptosis was moderate in 10 cases and severe in 4, while ophthalmoplegia was moderate in 4 patients and severe in 8 others. 10 patients were reported to have had 'visual disturbances'; most probably diplopia. 6 patients had moderate to severe facial palsies and there was moderate to severe paralysis of the tongue and palate in 9 patients. Paralysis of intercostal muscles was severe in 7 patients with 3 patients experiencing moderate diaphragmatic paralysis. Paralysis of the diaphragm was severe in only 1 patient. 7 patients had moderate, and 2 patients had severe, peripheral flaccid paralysis.

Campbell (1964) reports 'visual disturbances' in 26.9% of patients, noting both diplopia and blurred vision. Ptosis was regarded as the earliest sign of neurotoxicity and Campbell comments that many patients would go to sleep only to awake the next morning with severe facial palsy. Ptosis was often associated with either partial or complete ophthalmoplegia, and was observed in 75% of patients in this series. Campbell (1966) says that bites by death adders (*Acanthophis* spp.) produced either blurring of vision or an inability to see clearly, and that ocular and facial paralysis generally followed; however these signs were present in only 3 of the 15 patients. Campbell (1967a) states that 'muscular paralysis' was present in only 3 of 6 presumed Papuan taipan (*Oxyuranus scutellatus canni*) victims yet says that paralysis was present after 9 of 13 supposed Papuan blacksnake (*Pseudechis papuanus*) bites. Campbell's assertion that during the 1960's the Papuan blacksnake was the most common venomous snake in southern PNG and cause of the most snakebites is a fallacy that persists even today, and has resulted in many patients being treated incorrectly with inappropriate antivenoms.

Blasco & Hornabrook (1972) describe a fatal case of small-eyed snake (*Micropechis ikaheka*) envenoming marked by peripheral limb weakness, shortness of breath and profound respiratory failure with cyanosis. Warrell *et al* (1996) reviewed 6 cases of envenomation by this species (identity confirmed by EIA) and reported ptosis in 4 patients, bulbar paralysis in 3, and peripheral limb weakness in 3 cases. Ptosis was present between 6 and 12 hours post-bite. Two patients died of respiratory paralysis at 19 and 38 hours respectively. A patient from Papua developed dysarthria, facial paralysis and ataxic gait resulting in an inability to stand unassisted. Paralysis of the cranial muscles and intercostals was pronounced.

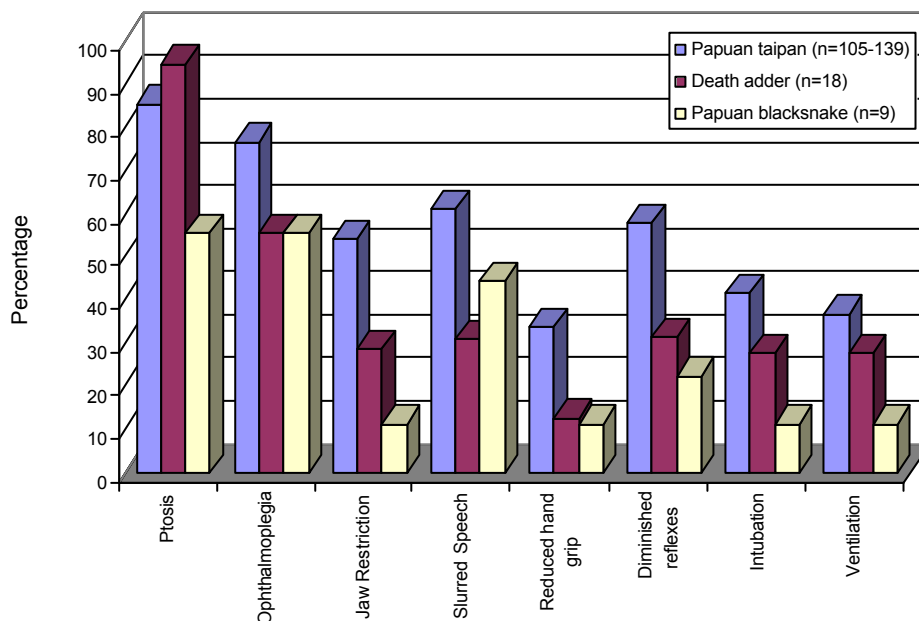
In a study of 32 cases of proven death adder (*Acanthophis* spp.) bites, Lalloo *et al* (1996) found that 18 developed symptoms and signs of envenomation. Ptosis was a symptom in 15/18 (88.2%) and a sign in 17/18 (94.4%). Other symptoms included dysphagia (29.4%), dysarthria (37.5%), diplopia (18.8%), and respiratory difficulty (20%). On examination there were signs that these patients had ophthalmoplegia (55.6%), slurring of speech (30.3%), jaw restriction (28.6%), diminished hand grip (31.3%) and reduced reflexes (12.5%). A total of 5 patients (27.8%) had to be intubated and ventilated, with the median ventilation time being 13

hours. The range of ventilation times was between 2 and 23.5 hours, considerably less than for victims of Papuan taipans (*Oxyuranus scutellatus canni*). Lalloo *et al* (1995a) reported that the median ventilation time after bites by Papuan taipans was 88 hours with a range of from 3 to 176 hours.

Hudson & Pomat (1988) reported ptosis as a symptom in 55% and as a sign in 53% of snakebite envenomations in Madang province. Dysphagia and dysarthria were symptoms in 31%, and 20% reported diplopia. Respiratory distress was a symptom in 9% of bites. Other signs included bulbar paralysis (17%) and external ophthalmoplegia (13%). Only 5% of patients subsequently developed respiratory failure. The only highly venomous species in the province are death adders (*Acanthophis* spp.) and small-eyed snakes (*Micropechis ikaheka*). Hudson (1988a) reports complete bilateral ptosis, ophthalmoplegia and paralysis of tongue, jaws and pharynx in a man bitten by a death adder. Peripheral limb weakness was present as well as paralysis of the intercostal muscles. Hudson (1988b) reports bilateral ptosis, ophthalmoplegia, diplopia, bulbar and peripheral paresis in two patients presumed to have been bitten by small-eyed snakes (*Micropechis ikaheka*) in Madang province. Respiratory distress was evident in both patients, and one of them, a 13 year old girl, eventually died from cardiorespiratory failure 42 hours post bite. Resolution of ptosis, bulbar paresis and limb weakness took more than 14 days in the other patient despite the use of a large quantity of antivenom.

The study of Papuan taipan (*Oxyuranus scutellatus canni*) envenomation by Lalloo *et al* (1995a) remains the most comprehensive investigation of neurotoxicity by this large and very dangerous snake. Of 166 patients with confirmed bites by taipans, 139 (83.7%) developed signs and symptoms of envenomation. Ptosis was present in 84/131 (64.1%) patients and was the most frequently reported symptom followed by dysarthria in 39/121 (32.2%) and dysphagia in 39/124 (31.5%). Diplopia was reported in 26/120 (21.7%) and 17/121 (14%) had dyspnoea; 41.7% of patients required intubation as paralysis deepened, and ventilation was required by 36.7% of envenomed patients. Progression to the point where intubation was required ranged from 3 to 55 hours with a median time point of 13.5 hours.

FIGURE 6: Comparison of clinical signs of neurotoxicity after the EIA-proven bites of three different species of highly venomous Papua New Guinean snakes. (SOURCES: Lalloo *et al*, 1994; Lalloo *et al*, 1995a; Lalloo *et al*, 1996)



Williams & Bal (2003) reported ptosis and dysphagia in all 9 patients with presumed Papuan taipan (*Oxyuranus scutellatus canni*) envenoming, 5 had diplopia, 6 had dysarthria, 5 had general bulbar paralysis, 4 had dyspnoea and 2 had peripheral limb weakness. Eight of the 9 patients died from respiratory failure. McGain *et al* (2004) reported neurotoxicity (one or more of ptosis, dysphagia or dyspnoea) in all 60 patients in their series who died from snakebite in the PMGH ICU between January 1992 and December 2001.

Brian & Vince (1987) reported ptosis (88%), drowsiness (56%), ophthalmoplegia (48%), respiratory weakness (42%), difficulty swallowing (40%), slurred speech (38%), limb weakness (27%) and jaw weakness (23%) in a series of 49 paediatric cases of snakebite admitted to PMGH between August 1981 and October 1984. Sixteen of these patients required subsequent intubation, although all 49 had received antivenom. In Trevett *et al*'s (1994a) examination of referral letters submitted with patients brought to PMGH from outlying health centres in Central and Gulf provinces, ptosis (either with or without concurrent ophthalmoplegia) was identified at the health centre in only 33% of cases, despite the fact that 82% of the patients had signs of ptosis/ophthalmoplegia on arrival at the hospital. Other signs and symptoms of neurotoxicity were not provided in the referral letters.

Williams *et al* (2003) reported symptoms and signs of neurotoxicity in 423 patients admitted to health centres in the Mekeo region between January 1997 and December 2001. Ptosis was reported in 68.6% of these cases. Other conditions were less frequently reported: ophthalmoplegia (2.8%), diplopia (21.7%), dysarthria (22.7%), dysphagia (41.4%), and dyspnoea (17.7%).

Other clinical effects of envenomation

Many of the victims of snakebite present at rural health centres with symptoms and signs that do not neatly fit into the three major clinical categories of coagulopathy, myotoxicity and neurotoxicity.

Campbell & Young (1961) note that transient tachycardia was an occasional feature of envenomation. Blasco & Hornabrook (1972) reports tachycardia during the terminal stages of small-eyed snake (*Micropechis ikaheka*) envenomation. Both tachycardia and bradycardia were observed in patients with proven envenoming by this species reported by Warrell *et al* (1996). One victim of death adder (*Acanthophis* spp.) envenoming from Western province presented to the rural health centre with a heart rate of 100 bpm, and subsequently developed both renal failure and a second-degree atrioventricular block (ventricular rate of 36 bpm). Laloo *et al* (1997) reported a single case of a death adder victim with septal T-wave inversion, as well as in a single case of New Guinean brown snake (*Pseudonaja cf. textilis*) envenomation. Laloo *et al* (1995a) found that 24.1% of Papuan taipan (*Oxyuranus scutellatus canni*) victims presented to hospital with bradycardia but were rarely hypotensive, and 52.2% of those tested had electrocardiograms that demonstrated abnormalities, particularly septal T-wave inversion (31.9%).

Tachycardia was an early feature in 5 fatal cases of presumed Papuan taipan (*Oxyuranus scutellatus canni*) envenomation reported by Williams & Bal (2003). Williams *et al* (2003) reported tachycardia in 20% of 564 snakebite patients from Mekeo for whom hourly blood pressure readings were available, while 19.8% had bradycardia, 24.1% were hypertensive and 25.5% were hypotensive. Campbell (1967a) notes that sudden collapse after snakebite is common following bites by Australian species. Laloo *et al* (1995a) reported sudden early collapse with transient loss of consciousness as a feature in 23% of Papuan taipan (*Oxyuranus scutellatus canni*) bites. McGain *et al* (2004) reported early collapse in 13.3% of the 60 fatal cases reported in the ICU at PMGH.

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Answers to ‘Can you pick the REAL Papuan blacksnake?’

- | | |
|---|-----------------------------|
| (A): D’Albert’s python (<i>Leiopython albertisi</i>) | Non-venomous |
| (B): Papuan taipan (<i>Oxyuranus scutellatus canni</i>) | Highly venomous & dangerous |
| (C): Brown-headed snake (<i>Furina tristis</i>) | Moderately venomous |
| (D): Papuan blacksnake (<i>Pseudechis papuanus</i>) | Highly venomous & dangerous |
| (E): Solomon’s coral snake (<i>Salomonelaps par</i>) | Moderately venomous |
| (F): Papuan taipan (<i>Oxyuranus scutellatus canni</i>) | Highly venomous & dangerous |
| (G): Papuan blacksnake (<i>Pseudechis papuanus</i>) | Highly venomous & dangerous |
| (H): Slatey-grey snake (<i>Stegonotus cucullatus</i>) | Non-venomous |