Southern African arrow poison recipes, their ingredients and implications for Stone Age archaeology

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ABSTRACT

Biochemical analyses of residues preserved on ethno-historical and archaeological artefacts increase our understanding of past indigenous knowledge systems. The interpretation of biochemical traces is, however, difficult. Problems that can hamper credible interpretations of ethno-historical or archaeological residues include incomplete knowledge about local natural products, limited published data about product applications, and overestimation of the abilities of the analytical techniques to make specific identifications. In an initial attempt to address some of the challenges, we discuss arrow poison as a case in point, and we provide complete, updated inventories of known southern African poison ingredients and recipes, suspected poisons, and the current state of knowledge about these toxins and their effects. We also suggest that discoveries of ancient arrow poison, and the technical steps involved in early toxicology, have the potential to indicate levels of human cognition.

KEY WORDS: biochemistry, cognition, hunting poisons, poison recipes, potential poisons.

In the ethno-historical literature of southern Africa, poisoned arrows are synonymous with San hunter-gatherers. Hunting, especially of large game, played a pivotal role in the social cohesion of such populations (e.g. Wiessner 2002). Due to the delicate nature of the reed-shafted arrows and lightweight bows (Fig. 1), a large animal shot with an arrow would be unlikely to succumb without the additional use of poison on the arrowhead (Marshall 1960; Guthrie 1983). The hunting aspect of Stone Age hunter-gatherer lifeways is effectively traceable through faunal and artefact analyses, and it is broadly accepted that throughout the Holocene (from ~12 000 years ago) people hunted with poisoned arrows in southern Africa (e.g. Wadley 1998). Weapon-delivery systems, such as hand-delivered spear hunting, and mechanical delivery systems, such as spearthrower-and-dart or bow-and-arrow hunting, can sometimes be distinguished deeper in time, using functional studies (e.g. Lombard & Phillipson 2010; Hardy et al. 2013). Such distinctions are important because evidence for the development of technologies such as bow hunting has the potential to highlight aspects of technological complexity and past cognition (e.g. Lombard & Haidle 2012; Williams et al. 2014). More subtle innovations in hunting systems, such as the introduction of poisons, also have great potential to inform us of past cognitive frameworks and the time depth of indigenous knowledge systems. Tracing such techno-behaviours through the Stone Age is not an easy task.

Others have suggested previously that early manifestations of microlithic stone tool assemblages in the Later Stone Age, and perhaps even in the Middle Stone Age, with and without backed segments, may have been designed to be used with poisons (e.g. Clark 1977; Ambrose 2002). The interpretation of bone and stone implements
as arrow components, from contexts dating to more than 60,000 years ago at Sibudu, KwaZulu-Natal, South Africa, is pushing back the claimed invention of bow hunting to the Middle Stone Age in southern Africa (Backwell et al. 2008; Lombard & Pargeter 2008; Wadley & Mohapi 2008; Lombard & Phillipson 2010; Bradfield & Lombard 2011; Lombard 2011). Bone points, morphologically akin to those used as arrowheads by recent hunter-gatherers, have been found at White Paintings Shelter, Botswana, dating to between 35,000 and 37,000 years ago, leading the excavators to suppose a great antiquity for the use of poison at the site (Robbins et al. 2012). Similar suggestions have been made for bone points from Border Cave dating from ~43,000 years ago, interpreted as originating from early Later Stone Age contexts (d’Errico et al. 2012a). Together with the above archaeological evidence, the recent publication of chemically detected toxins using gas chromatography coupled with mass spectrometry (GC-MS) on residues from a purported 24,000-year-old poison applicator at Border Cave, KwaZulu-Natal (d’Errico et al. 2012a), has opened up questions about the potential antiquity of bow hunting, the use of arrow poisons, and the variety of ingredients that may have been used in the past.

The parent toxin on the Border Cave applicator was identified as ricin, originating from *Ricinus communis* beans (castor bean plant) (d’Errico et al. 2012a). Because ricinoleic acid and ricinelaidic acid (the components suggested as evidence of ricin poison) are the main components of castor oil and not necessarily poisonous themselves, and because castor plant products are widely recorded for their medicinal applications, Evans (2012) questioned the interpretation of the residue as arrow poison. He also pointed out that castor oil is known for hide preparation in the rural areas surrounding Border Cave (Evans 2012, see also the response by d’Errico et al. 2012b). Both interpretations could
be correct, because most ethnographically-known arrow poison ingredients also have medicinal and/or other practical applications (e.g. Neuwinger 1996). The dose often determines whether a product is medicine or poison. Furthermore, plant-based toxins are important natural sources for new drug discoveries, providing knowledge about biologically active molecules for pharmacological research (e.g. Philippe & Angenot 2005).

While ricin from castor beans might be effective as an arrow poison (see Appendix B), we could not find any southern African ethnographic reference to such use in any context. What is more, there is uncertainty about whether the castor bean plant (usually not considered indigenous to southern Africa) was present on the landscape 24 000 years ago. The plant is seen as indigenous to northeastern Africa and India, but has become a weed in southern Africa and many other parts of the globe (Van Wyk et al. 2002). It is also important to note that *Abrus precatorius* (crab’s eye creeper), indigenous to South Africa in the Eastern Cape, KwaZulu-Natal, Limpopo and Mpumalanga, has been recorded as an arrow poison ingredient used in eastern and southwestern Africa (Watt & Breyer-Brandwijk 1962). The plant seed contains the toxin abrin (A and C), which is very similar in structure and properties to ricin (Wei et al. 1974; Van Wyk et al. 2002; Dickers et al. 2003). Working with unknown, decayed material from archaeological contexts where analysts have to rely on the oxidative by-products, rather than the original compounds, further increases difficulty in the accurate identification of substances. The Border Cave results can thus be accepted as early evidence for the use of poison, but accurately pinpointing its source may not be straightforward.

It is sometimes thought that, thanks to more than a century’s worth of ethno-historical and anthropological recording, almost everything is known about San hunter-gatherer arrow poison ingredients. The Border Cave evidence suggests, however, that there are likely to be more toxins on archaeological material that has not yet been chemically analysed. Sooner, rather than later, the traditional use and associated indigenous knowledge of such substances will disappear, so there is a pressing need to study as many hunter-gatherer poisons as possible for their active constituents (e.g. Neuwinger 1998; Roberts & Wink 1998). In this paper we reflect on the use of poison recipes (compound/synthetic poisons) and touch on their potential cognitive implications. We present information on known southern African hunter-gatherer poison recipes into which multiple ingredients were mixed. Our intention is for these data, and those contained in the appendices, to provide a resource for future biochemical testing. The appendices build on the extensive publications on such poisons (e.g. Schapera 1925; Watt & Breyer-Brandwijk 1962; Shaw et al. 1963; Bisset 1989; Neuwinger 1996; Philippe & Angenot 2005), but we have added new/additional data and observations to provide an up-to-date, easily accessible archive for southern African hunter-gatherer arrow poisons.

ETHNO-HISTORICAL ARROW POISON RECIPES

Although considerable progress has been made in the understanding of the biochemical properties of ‘raw’ and isolated arrow poison ingredients since the analysis/synthesis published by Shaw and colleagues (1963; see also Bisset 1989, 1991; Neuwinger 1996, 1998; Philippe & Angenot 2005), much remains unknown and not all sources of the poison ingredients have been fully explored with state-of-the-art biochemical detection techniques. In Appendix A we list all southern African hunter-gatherer arrow poison
ingredients that we were able to trace in the published record, summarising what is known about their toxicity and the substances responsible for the toxic effects. We also provide a list of potential toxins (Appendix B). These are not known from San hunter-gatherer contexts, but were sometimes used by non-San hunters of the region. The rationale for this additional list is that almost no ethno-historical records exist for suitable San hunter-gatherer poisons used in areas other than the dry western parts of southern Africa. Some non-San records thus provide potential sources for poisons used by bow hunters in the savannah and/or tropical regions of the subcontinent. Almost all the poisonous plants in our appendices also have medicinal applications (e.g. Neuwinger 1996; Van Wyk & Gerricke 2000; Philippe & Angenot 2005). The preparation, dosage and administration of the plant extracts largely determine their potential efficacy as medicine or their harmfulness as poison.

The contents of our appendices demonstrate that the indigenous knowledge systems behind the use of plant- and/or animal-based poisons are extensive. Once substances are treated, for example with heat, and once different ingredients are mixed with the aim of producing synthetic poisons, much deeper knowledge and skill sets are evident. Poison recipes used historically by southern African hunter-gatherers varied by region, season, and availability of ingredients and prey type. For example, *Diamphidia* arrow poison is often mixed with *Sansevieria* juice in the Kalahari (Fig. 2). In some instances animal-based ingredients form the basis of arrow poisons (e.g. De la Harpe et al. 1983;
Dalay 1998), but generally, plant extracts containing alkaloids, terpenoids and glycosides are the main toxic compounds for arrow poisons world-wide (Philipe & Angenot 2005: 85). Many ingredients can be added for a variety of reasons, such as enabling thickening, adhesion and/or boosting toxicity; other motivations for additives could include magical purposes or to conceal the real composition of the poison (Philipe & Angenot 2005: 85). The effects of preparation processes such as heating, and the interplay amongst ingredients mixed into compound poisons, are poorly understood. For example, in any given recipe, does the treatment and/or mixing of different ingredients increase the toxicity, longevity and/or adhesive qualities of the poison? Which ingredients and preparation processes are aimed at altering the nature of the poison? Or, were some elements in the mix purely as a result of social convention, perhaps adding little or nothing to the functionality or preservation of the poison?

The possible use of heat-treated and/or synthetic poisons during the Stone Age also poses a significant challenge for the biochemical analysis of archaeological residues because it may alter signatures based on the analysis of single ‘raw’ ingredients (see Appendix A & B). In Table 1 we present all known San arrow-poison treatments and recipes with their expected toxins and molecular compositions, as far as we were able to access these data and the regions or groups known to have used them. In Figure 3 we present some of the species that have been implicated in hunting poisons among the Hei|om and Ju|wasi. The information demonstrates the potential complexities for future biochemical analyses of archaeological residues. By nature, the hunt for archaeological poison use must be a cross-disciplinary effort, but most biochemists working on archaeological samples, and researchers not intimately familiar with local ethnography on arrow poisons, might not be aware of the body of existing ethnohistorical knowledge represented here and in our appendices.

ARROW POISONS, INDIGENOUS KNOWLEDGE SYSTEMS AND COGNITION

Different toxins may have different effects, depending on the size and habits of the prey. When hunting for subsistence, care must be taken over the specific poison used for particular animals. For example, fowl cannot be hunted using coniine, as this toxin will contaminate the meat (Lopez et al. 1999). In contrast, a mammal poisoned with coniine can be eaten, but the milk will be contaminated (Lopez et al. 1999). Modern hunter-gatherers seem to have been aware of such variability in the properties of hunting poisons; for example, in the southern African context, both Stow (1905) and Watt and Breyer-Brandwijk (1962) state that different poisons were used to hunt different animals. The use of poisoned arrows thus represents multi-layered and complex indigenous knowledge systems.

A further aim of collating what is known about San hunter-gatherer arrow poison preparation processes and recipes is to explore what these are able to reveal about cognition. If we understand the attributes of cognition inherent in these techno-behaviours in the context of current or recent bow hunters, the insights can be used to trace similar cognitive attributes through deep time. One way of effecting transformation, an important attribute that points to complex cognition (Wadley 2013), is to combine individual ingredients so that they are irreversibly altered, and a novel product is created. The mixing of compound poisons seems to be an obvious example.
TABLE 1

Ethno-historically known arrow-poison preparation processes and recipes (see Appendix A for discussion of single components). Note that in this table we prefer the name Juwasi to the older version !Kung, but that the latter appellation is used by our sources.

<table>
<thead>
<tr>
<th>Recorded poison preparation and/or recipe</th>
<th>Expected toxins and their known molecular composition</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 <em>Acokanthera</em> poison is prepared by boiling cuttings of the plant for up to 10 hours until a viscid consistency is reached (Schapera 1925; Neuwinger 1996). <em>Acokanthera</em>-based poison was used in the vicinity of Tulbagh to hunt large animals (Thunberg 1986) as well as by the Kalahari Juwasi and Eastern Cape San (Lebzelter 1996).</td>
<td>Acovenoside A (C_{30}H_{46}O_{9}); ouabain (C_{29}H_{44}O_{12})</td>
<td>Boiled</td>
</tr>
<tr>
<td>2 <em>Acokanthera</em> mixed with <em>Euphorbia</em> sap (Watt &amp; Breyer-Brandwijk 1962).</td>
<td>Acovenoside A (C_{30}H_{46}O_{9}); diterpene (C_{20}H_{32}); ingenol (C_{20}H_{28}O_{5}); ouabain (C_{29}H_{44}O_{12}); phorbol (C_{20}H_{28}O_{6})</td>
<td>Boiled</td>
</tr>
<tr>
<td>3 <em>Acokanthera</em> is prepared in the same way as 1 above, but is then mixed with dehydrated snake venom, <em>Euphorbia</em> sap and/or <em>Boophane</em> sap (Dorman 1916 cited in Schapera 1925; Thunberg 1986).</td>
<td>Acovenoside A (C_{30}H_{46}O_{9}); diterpene (C_{20}H_{32}); ingenol (C_{20}H_{28}O_{5}); lectins; ouabain (C_{29}H_{44}O_{12}); phorbol (C_{20}H_{28}O_{6}); phospholipase A; serin proteases</td>
<td>Boiled</td>
</tr>
<tr>
<td>4 In a non-San context, Neuwinger (1996) reports <em>Annona chrysophylla</em> as an ingredient in some <em>Acokanthera</em> poisons among the Lobedu and Venda. No method of preparation is given.</td>
<td>Acovenoside A (C_{30}H_{46}O_{9}); anonaine (C_{17}H_{15}NO_{2}); ioboldine (C_{19}H_{21}NO_{4}); liriodenine (C_{17}H_{9}NO_{3}); ouabain (C_{29}H_{44}O_{12})</td>
<td>Sundried</td>
</tr>
<tr>
<td>5 The Heikom, Herero and Nama of Namibia made holes or cuts in the stems of <em>Adenium</em>, gathered the sap in a vessel and dried it in the sun. The dried substance was mixed with saliva and applied to the arrow (Shaw et al. 1963).</td>
<td>Echujine; hongheloside B (C_{35}H_{56}O_{14}); obobioside B (C_{38}H_{58}O_{15}); somaline; tetraphyllin B (C_{12}H_{17}NO_{7})</td>
<td>Boiled, sometimes with a little water</td>
</tr>
<tr>
<td>6 The flowering bulb of <em>Adenium</em> is dug up and the sap is squeezed out, and boiled to condense it. A little water is sometimes added during the boiling, and when cool the syrup is applied to the arrow (Shaw et al. 1963).</td>
<td>Echujine; hongheloside B (C_{35}H_{56}O_{14}); obobioside B (C_{38}H_{58}O_{15}); somaline; tetraphyllin B (C_{12}H_{17}NO_{7})</td>
<td>Boiled over fire</td>
</tr>
<tr>
<td>7 Thick branches and roots of <em>Adenium</em> are cut and heated over a fire. The thick liquid that oozes out is smeared onto the arrowhead. Additives that might be included are <em>Euphorbia</em> latex, <em>Spirostachys africana</em> sap and <em>Aloe</em> species sap (Neuwinger 1996). <em>Adenium</em> species are used widely throughout central and northern Namibia (Fourie 1926; Neuwinger 1996). <em>Spirostachys africana</em> sap is reported as an additive to the <em>Adenium</em> poison of the Hei</td>
<td>om (Fourie 1926).</td>
<td>A-coniceine (C_{8}H_{15}N); conhydrine (C_{8}H_{17}NO)</td>
</tr>
</tbody>
</table>
Nadler (2005) reports that the Ju|wasi mixed *Adenium* stem and/or root sap with *Diamphidia* entrails. The *Boophane* bulb is cut transversely and the fluid extracted. The thick fluid is kept in the sun until it achieves a wax-like consistency, which is kept until needed. The resulting substance is added to *Euphorbia* latex and dehydrated, powdered snake venom (Livingston 1857; Schapera 1925; Lichtenstein 1930; Goodwin 1945). Occasionally dried, powdered spiders (Farini 1973), and/or *Acokanthera* sap were added (Dornan 1925; Hall & Whitehead 1927).

**TABLE 1 (continued)**

Ethno-historically known arrow-poison preparation processes and recipes (see Appendix A for discussion of single components). Note that in this table we prefer the name Ju|wasi to the older version |Kung, but that the latter appellation is used by our sources.

<table>
<thead>
<tr>
<th><strong>Region</strong></th>
<th><strong>Process</strong></th>
<th><strong>Ingredients</strong></th>
<th><strong>Notes</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>Nadler (2005) reports that the Ju</td>
<td>wasi mixed <em>Adenium</em> stem and/or root sap with <em>Diamphidia</em> entrails.</td>
<td>Diamphotoxin; echujine; hongheloside B (<strong>C_{36}H_{56}O_{14}</strong>); obobioside B (<strong>C_{38}H_{58}O_{15}</strong>); somaline; tetraphyllin B (<strong>C_{12}H_{17}NO_{7}</strong>).</td>
</tr>
<tr>
<td>9</td>
<td>The <em>Boophane</em> bulb is cut transversely and the fluid extracted. The thick fluid is kept in the sun until it achieves a wax-like consistency, which is kept until needed. The resulting substance is added to <em>Euphorbia</em> latex and dehydrated, powdered snake venom (Livingston 1857; Schapera 1925; Lichtenstein 1930; Goodwin 1945). Occasionally dried, powdered spiders (Farini 1973), and/or <em>Acokanthera</em> sap were added (Dornan 1925; Hall &amp; Whitehead 1927).</td>
<td>L-atrotoxin; buphandrine; buphanine (<strong>C_{18}H_{21}NO_{4}</strong>); crinamidine; crinamine (<strong>C_{17}H_{19}NO_{4}</strong>); distichine; diterpene (<strong>C_{20}H_{32}</strong>); haemanthamine (<strong>C_{17}H_{19}NO_{4}</strong>); indolelactic acid-spermine; ingenol <strong>C_{20}H_{28}O_{5}</strong>; lectins; lycorine (<strong>C_{15}H_{18}O_{6}</strong>); phorbol (<strong>C_{20}H_{28}O_{6}</strong>); phospholipase A; serin proteases.</td>
<td>Sun thickened</td>
</tr>
<tr>
<td>10</td>
<td>Among the tribes of the Namib and Kalahari the white milky latex of <em>Euphorbia</em> is left in the sun to thicken and applied directly onto the arrow (Schapera 1925; Neuwinger 1996).</td>
<td>Diterpene (<strong>C_{20}H_{32}</strong>); ingenol (<strong>C_{20}H_{28}O_{6}</strong>); phorbol (<strong>C_{20}H_{28}O_{6}</strong>).</td>
<td>Dried and powdered</td>
</tr>
<tr>
<td>11</td>
<td>Among the tribes of the Orange River <em>Euphorbia</em> latex is mixed with the entrails of a freshly squeezed <em>Diamphidia</em> beetle and then left to congeal in the sun, after which the mixture is smeared onto arrows (Paterson 1789; Wikar 1779 cited in Mossop 1935).</td>
<td>Diamphotoxin; diterpene (<strong>C_{20}H_{32}</strong>); ingenol (<strong>C_{20}H_{28}O_{6}</strong>); phorbol (<strong>C_{20}H_{28}O_{6}</strong>).</td>
<td>Sun congealed</td>
</tr>
<tr>
<td>12</td>
<td>Among the Herero and Hei</td>
<td>om <em>Euphorbia</em> latex is dried and powdered and added to the juice of an <em>Adenium</em> species (Neuwinger 1996).</td>
<td>Diterpene (<strong>C_{20}H_{32}</strong>); echujine; hongheloside B (<strong>C_{36}H_{56}O_{14}</strong>); ingenol (<strong>C_{20}H_{28}O_{6}</strong>); obobioside B (<strong>C_{38}H_{58}O_{15}</strong>); phorbol (<strong>C_{20}H_{28}O_{6}</strong>); somaline; tetraphyllin B (<strong>C_{12}H_{17}NO_{7}</strong>).</td>
</tr>
<tr>
<td>13</td>
<td>The fruit and seeds of <em>Hyaenanche</em> are dried, powdered and mixed with <em>Euphorbia</em> latex (Watt &amp; Breyer-Brandwijk 1962).</td>
<td>Diterpene (<strong>C_{20}H_{32}</strong>); ingenol (<strong>C_{20}H_{28}O_{6}</strong>); phorbol (<strong>C_{20}H_{28}O_{6}</strong>); mellitoxin (<strong>C_{15}H_{18}O_{6}</strong>); urushiol III (<strong>C_{15}H_{18}O_{6}</strong>); isodihydrohyaenanchine.</td>
<td>Dried and powdered</td>
</tr>
<tr>
<td>14</td>
<td>The method of preparation of <em>Strophanthus</em> poison in southern Africa differs from methods used farther north. In southern Mozambique the seeds are crushed, mixed with saliva to a paste and exposed to sunlight for several hours, after which time the resulting paste is applied to the arrows (Neuwinger 1996).</td>
<td>Christyoside (<strong>C_{20}H_{28}O_{5}</strong>); cyamin (<strong>C_{16}H_{17}O_{4}</strong>); k-strophanthin; ouabain (<strong>C_{20}H_{14}O_{3}</strong>); strophanthidin.</td>
<td>Exposed to sunlight</td>
</tr>
</tbody>
</table>
15 The Hei||om and Ju||wasi northeast of Grootfontein, Namibia, use a *Strychnos* species, together with *Euphorbia* and *Boophane*, as additives to snake venom and *Diamphidia* poison (Hall & Whitehead 1927). The mixture is boiled for 10 minutes in a hollow stone into which the poison maker frequently spits during the intervals while chanting.

| 15 | Akagerine (C_{20}H_{24}N_{2}O_{2}); 10-hydroxyakagerine (C_{20}H_{24}N_{2}O_{3}); buphanine (C_{18}H_{21}NO_{4}); crinamine (C_{17}H_{19}NO_{4}); C-toxiferine I (C_{40}H_{46}N_{4}O_{2}); ingenol (C_{20}H_{28}O_{5}); lycorine (C_{26}H_{30}NO_{5}); phorbol (C_{29}H_{28}O_{7}) | Boiled |
| 16 | Catechin-tannin (C_{15}H_{14}O_{6}); haemolytic saponins; gypsogenin (C_{30}H_{46}O_{4}); kaempferol (C_{15}H_{10}O_{6}); O-acetyloleanolic acid (C_{32}H_{50}O_{4}); oleanolic acid (C_{30}H_{48}O_{3}) | Roasted |
| 17 | Catechin-tannin (C_{15}H_{14}O_{6}); diamphotoxin; gypsogenin (C_{30}H_{46}O_{4}); haemolytic saponin kaempferol (C_{15}H_{10}O_{6}); O-acetyloleanolic acid (C_{32}H_{50}O_{4}); oleanolic acid (C_{30}H_{48}O_{3}) | Roasted |
| 18 | Catechin-tannin (C_{15}H_{14}O_{6}); diamphotoxin; gypsogenin (C_{30}H_{46}O_{4}); kaempferol (C_{15}H_{10}O_{6}); L-cystein (C_{3}H_{7}NO_{2}S); O-acetyloleanolic acid (C_{32}H_{50}O_{4}); oleanolic acid (C_{30}H_{48}O_{3}); pipecolinic acid (C_{6}H_{11}NO_{2}); saponins | Heated |
| 19 | Diamphotoxin; saponins | Diamphotoxin; citrullin (C_{15}H_{11}N_{2}O_{3}); cucurbitacin E (C_{25}H_{40}O_{2}) | Sundried |
| 20 | The *Diamphidia* grub is dried in the sun and powdered. To the powder is added the partially masticated juice of an *Acacia* or the liquid from the heated roots of a *Citrillus* species (Schapera 1925; Bleek 1928; Shaw et al. 1963). | Diamphotoxin; citrullin (C_{15}H_{11}N_{2}O_{3}); cucurbitacin E (C_{25}H_{40}O_{2}) | Heat treatment of *Cucumis beptadactylus* root over a fire |
| 21 | The Auin hunter-gatherers of the Kaukau area in northern Namibia mixed the dried and powdered *Diamphidia* grubs with the root juice of the *Cucumis beptadactylus* (Kaufmann 1910 in Neuwinger 1996). The root of a *C. beptadactylus* was heated over a fire and the juice squeezed out. | Diamphotoxin; cucurbitacin B, D, G and H |
22 The Ju|wasi of the Nyae-Nyae area of Namibia are reported to have mixed the Diamphidia larvae with the juice of Solanum incanum or Solanum kwebense (Neuwinger 1996; Nadler 2005). No method of preparation is given.

Diamphotoxin; saponines; solasonine (C_{45}H_{73}NO_{16}); solmargine (C_{45}H_{73}NO_{15})

23 The Ju|wasi roast the root of a Protasparagus excavialis over a fire and mix the juice with the Diamphidia grub entrails (Neuwinger 1996).

Diamphotoxin (we could not find further information on the toxin/s associated with Protasparagus excavialis) Roasted

24 The crushed Diamphidia larvae is mixed with Protasparagus excavialis and the bulb juice of Urginea sanguinea by the Kxoe of Namibia. The mixture is concentrated by heating over a fire (Köhler 1973 cited in Neuwinger 1996).

Diamphotoxin; proscillaridin A (C_{40}H_{32}O_{8}); physodine A (C_{31}H_{44}O_{10}); scillaren A (C_{56}H_{32}O_{13}); urginin (C_{42}H_{62}O_{13}) Heating over a fire

25 The Gwi and Ju|wasi of Namibia mix the juice of a Terminalia sericea with the body contents of the Diamphidia grub. A twig of the T. sericea is chewed and the resulting liquid spat into the squeezed body contents of the grub (Neuwinger 1996). Occasionally the resin from Swartzia madagascariensis pods is added (Neuwinger 1996).

Diamphotoxin; resveratrol 3-O-rutinoside; sericoside (C_{36}H_{58}O_{11}); sericic acid (C_{30}H_{48}O_{6})

26 In central Botswana and eastern Namibia the fresh or powdered innards of the Diamphidia grub are mixed with the masticated bark and sap of an Acacia mellifera (Neuwinger 1996).

Diamphotoxin; L-cystein (C_{7}H_{11}NO_{2}S); pipercolinic acid (C_{6}H_{11}NO_{3}); saponins Powdered

27 Among the Ju|wasi in the Tsumke region of Namibia the Diamphidia grub is mixed with the heated juice of a Protasparagus excavialis, Sanseviera aethiopica and the fresh fruit pulp of Swartzia madagascariensis (Neuwinger 1996).

Diamphotoxin; saponins Heated

28 The poison glands of snakes such as Naja nivea (Cape cobra), Bitis arietans (puff adder) and Dendroaspis polyepics (black mamba) are removed, dried and powdered, to which is added the juice of Euphorbia or Aokanthera. The mixture is boiled until it reaches the consistency of thick jelly and obtains a reddish-brown colour before being applied to an arrow (Stow 1905; Dornan 1916 cited in Schapera 1925).

Acovenoside A (C_{30}H_{46}O_{9}); lectins ouabain (C_{29}H_{44}O_{12}); phospholipase A; serin proteases Dried and powdered, boiled after being mixed with other ingredients
Fig. 3: Potential range of ingredients described for recipe no. 15 in Table 1. Clockwise from the top left: *Strychnos pungens* tree (photograph by LW), *Strychnos pungens* fruit (photograph by LW), *Boophane disticha* (photograph by LW), *Diampidia* grub capsules collected for poison making (photograph by Estelle Oosthuysen© and reproduced with her permission), *Euphorbia ingens* (photograph by ML).
There are already two examples of this type of technology in the Middle Stone Age of South Africa. First, a variety of recipes for making compound adhesives has been suggested to explain the combinations of plant, animal and mineral products that were found on >70 000–58 000-year-old Middle Stone Age stone tools from Sibudu (Wadley 2005; Lombard 2006, 2007; Wadley et al. 2009). Secondly, a compound mixture, interpreted as paint, was found in two abalone shells in a 100 000-year-old occupation at Blombos Cave (Henshilwood et al. 2011). Replications (e.g. Wadley et al. 2009) imply that the skilled workers who made compound adhesives or paints must have mentally abstracted the individual attributes of ingredients before they could successfully combine them and that they needed to multitask during the tasks.

Blending poisonous ingredients to create arrow poison is no different from the processing of compound adhesive or paint, and we suggest that comparable principles were followed, using similar mental processes. The toxicity of snake venom from a single species is not constant (see Appendix A) and the toxicity of particular plants may vary by season or region. For example, the pH of *Acacia karroo* gum varies from tree to tree: samples from different trees were found to have pH readings between 3 and 4.4 (the first pH being 10 times more acidic than the second) (Wadley et al. 2009). When gum is added to powdered ochre when making compound adhesive, the merged product becomes more basic (Wadley et al. 2009), and it seems likely that similar reactions take place when plant or animal toxins are blended with gums or other plant products. However, the purpose of blends may not always be to increase toxicity; some ingredients may be added in order to alter the texture for ease of application, or to stabilise the product. *Acacia* gum, for example, is sometimes used commercially to stabilise compound food items to prevent their ingredients from separating.

Today’s hunter-gatherers may not have a formal understanding of chemistry or chemical reactions, but they have an indigenous knowledge system that enables them to use plant and animal extracts effectively for medicines or poisons. Using Wynn and Coolidge’s (2007) terminology, this is ‘procedural knowledge’. This type of knowledge also incorporates ethology, that is, understanding of prey ecology and behaviour. Ethology is required for successful meat-procurement, whether this be through spear hunting, setting of traps or snares, or bow-and-arrow hunting. Today’s San hunters use all these methods and we suggest that they may have ancient origins. Each hunting technique has cognitive correlates. Out-of-sight, long-distance action involving response inhibition, such as setting snares, seems to be a convincing proxy for complex cognition (Wadley 2010). The action takes place out of sight of the hunter. The use of a poisoned arrow is both similar to and different from the setting of a snare. The combinations of active and passive meat-getting strategies, and the presence of visible and invisible stages of the hunt, make the use of poisoned arrows a more complex behaviour than either snaring or hunting alone. It is similar to using a snare in the sense that the poison kills the animal out-of-sight of the hunter. It is different from a snare in that the arrow that is shot to transmit the poison is an active intervention by the hunter.

**DISCUSSION AND CONCLUSION**

Southern Africa has a wide variety of poisonous plants and animals that are suitable for use as ingredients in arrow poisons. Yet, only a fraction of these species has been recorded in this context or has been analysed for phytochemistry. Some hunter-
gatherers in the Middle Stone Age had an intimate knowledge of the medicinal and/or insecticidal properties of plants (Wadley et al. 2011; Wadley 2013), and macro-botanical remains of plants with known medical and poisonous properties (e.g. Boophane disticha, Swartzia madagascariensis and Strychnos sp.) have been found in several Later Stone Age archaeological contexts (e.g. Fagan & Van Noten 1971; Wadley 1987; Binneman 1999; Deacon & Deacon 1999). Such discoveries bear testament to the antiquity of the use of poisonous plants in hunter-gatherer societies. Southern Africa is also the location of possibly the oldest known evidence for bow hunting in the form of bone and stone arrow tips dating between about 65 000 and 35 000 years old—much earlier than previously thought. Chemical tests of residues found at Border Cave have provided indications for the use of toxic plant species not previously known in the context of southern African arrow poisons. As a result, new questions are arising regarding not only the antiquity of poison use, but also the variety of ingredients and recipes used in the past, especially in regions for which no ethnographic record exists.

Knowledge of the toxic effects of plants has a great antiquity in southern Africa. At Sibudu Cave in KwaZulu-Natal, the inhabitants were repelling insects with the mildly toxic leaves of Cryptocarya woodii (Cape quince) at 77 000 years ago, and were burning the wood of Spirrostachys africana (tamboti), which can release toxic fumes, by 58 000 years ago (Wadley et al. 2011; Wadley 2013). The latter is also known to have been used as a San arrow-poison ingredient in recent times (Neuwinger 1996; Nadler 2005; Appendix A), so tamboti may have had multiple roles in the past. There are many other plants in southern Africa that are known to have lethal toxins but which are not known to have been used as hunting poisons in the past—whether for want of use or want of adequate ethnographic information in certain areas of the subcontinent, we do not know. For instance, piperidine alkaloids are lethal neurotoxins that are well known in the context of South American poison-dart hunting, where they are sourced from Dendrobatidae frogs (Dalay 1998). Approximately 16 % of African Aloe species also contain such alkaloids, some of which have been observed as hunting poisons in East Africa (Neuwinger 1996). In southern Africa, lethal piperidine alkaloids are present in several indigenous Aloe species such as A. globuligemma, A. krapholiana and A. gariepensis (Reynolds 2005). Additionally, these alkaloids are structurally identical to coniine found in poison hemlock (Conium sp.), and there are several indigenous varieties of Conium including C. chaerophyllum, C. fontanum and C. Sphaerocarpum that contain toxic piperidine alkaloids (Hilliard & Burtt 1985). Thus, in addition to the biochemical identification, we argue that in-depth knowledge of plant distribution patterns, their toxic properties and ethno-historical records is necessary to generate robust interpretations of ancient poison use. Also, many animal toxins, such as snake venom (Appendix A), are generally far more complex than plant toxins. Venoms usually consist of many polypeptide proteins and an assortment of other compounds, making chemical identification from an unknown source, like an arrow, challenging. Some toxins, for example coniine, are found in different and unrelated species, such as Conium maculatum and Aloe globuligemma (Reynolds 2005).

Thus far, the use of poison for hunting purposes has not been reported for any hominin species but our own. Technologies that can be used to explore the potential cognitive and behavioural uniqueness of our own species/subspecies, and/or intra-species trends in cognitive and behavioural evolution, are therefore important resources
The ability to mix toxins effectively implies long attention spans, response inhibition, the capacity for novel, sustained multilevel operations, the use of abstract thought, and the ability to plan the assembly of ingredients as well as complex action sequences. In short, the mixing of compound poisons implies attributes of cognition that are like those we associate with people today. When analysing the steps required to implement a technical strategy such as the creation of a compound poison, we argue that some of the steps may have been impossible without the attributes of complex cognition. Complex cognition is taken for granted amongst people who mix poisons today. However, the cognitive implications of making compound poisons become especially significant if and when such compounds are discovered on weaponry from many thousands of years ago.

Thus, for a range of reasons, concerted cross-disciplinary efforts to detect and identify Stone Age arrow poisons with state-of-the-art techniques are likely to prove a useful quest. In our appendices we have provided a list of known and other possible poison ingredients, both plant and animal, and what is currently known of their biochemistry. All the species listed are suitable hunting poisons and, in most cases, have served this purpose among groups in different parts of Africa (hunter-gatherer or otherwise). Future biochemical work on ethno-historical and archaeological arrow poisons will extend the range of known poison ingredients, and we predict that future biochemical ‘fingerprinting’ of the ingredients presented in our appendices might lead to the discovery of new toxins and/or chemical compounds. Such new knowledge might even lead to the (re)discovery of medicinal properties associated with some of our indigenous plants, and will certainly enhance our understanding of past indigenous knowledge systems and their associated behaviours and cognitive skills.

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Appendix A

Arrow poison ingredients known to have been used by hunter-gatherers in southern Africa

ANIMAL POISON INGREDIENTS

Snake venom
Venom from a variety of snakes, including *Naja nivea* (Cape cobra), *Bitis arietans* (puff adder) and *Dendroaspis polylepis* (black mamba), has been used as an arrow poison ingredient (e.g. Dornan 1925; Schapera 1925; Thunberg 1986; Nadler 2005). Most venom toxins are resistant molecules that can survive boiling and exposure to weak acidic solution, and they are only denatured by urea and strong alkali resulting from desulfurisation (Karlsson 1979). When bitten by a snake, the San are reported to have urinated on the wound in an attempt to destroy the venom (Arbousset cited in Schapera 1927). Although the venom’s protein structure breaks down when exposed to air, this seems to have no adverse effect on the toxicity of the venom (Jesupret et al. 2014). Indeed, snake venom can remain pharmacologically active for at least up to 80 years (e.g. Shaw et al. 1963; Jesupret et al. 2014).

Snake venoms are complex mixtures containing various neurotoxins, cardiotoxins, cytotoxins and coagulation factors acting alone or synergistically (Karlsson 1979). The exact number of compounds that venom contains is unknown, but most constituents are proteins of low molecular weight such as peptides, nucleosides and metal ions (Karlsson 1979; Currier et al. 2010). Three types of protein-based toxins that are heat-resistant and act as depolarising agents have been identified in *N. nivea* (Botes et al. 1971; Earl & Excell 1972), but these proteins have not been named. The venom protein’s toxicity is thought to be dependent on the disulfide bonds (Botes 1974), and is dependent also on environmental factors. For instance, the venom of the Cape cobra is more potent in the Northern Cape and Namibia than it is in the Western Cape (B. Muller pers. comm. 2014).

*B. arietans* has extremely effective haemorrhagic and cytotoxic venom. Currier and colleagues (2010) identified, among other compounds, serine proteases, disintegrins and C-type lectins in puff adder venom. The venom of *D. polylepis* contains a 60-amino acid peptide chain called calciseptine. The peptide is a smooth muscle relaxant and inhibits heart contractions (De Weille et al. 1991). Three additional toxins, isolated from the venom of the black mamba, are called muscarinic toxins. All of these toxins have four disulphide bonds and 65 or 66 amino acids, and are similar to many other snake venom components, such as neurotoxins, cardiotoxins, and fasciculins (Jolkkonen et al. 1995).

Araneae
Trapdoor spiders of the Ctenizidae and Migidae families are also known to have been used in arrow poisons (Livingstone 1857; Stow 1905; Schapera 1925; Nadler 2005). Whole spiders were ground up and mixed with other ingredients (Schapera 1925). The trapdoor spider venom is, however, not considered to have contributed significantly to the efficacy of the poison other than providing a local irritant (Shaw et al. 1963; Bisset 1989; Nadler 2005).

Similar to other animal venoms, spider venomss are heterogeneous between and within species. They consist of complex mixtures of bioactive and inactive substances,
with the key constituents in the venom represented by proteins, polypeptides and polyamine neurotoxins, enzymes, nucleic acids, free amino acids, monoamines and inorganic salts (Rash & Hodgson 2002). Trapdoor spiders, and their cousin, the baboon spider, are widespread in tropical and sub-tropical areas of the world, but only one species (Harpactirella lightfooti) contains a neurotoxin that can be considered harmful (Tipton & Dajus 1994; Dippenaar-Schoeman 2002). H. lightfooti is confined to the south-western Cape of South Africa (Bowles & Swaby 2006). The primary toxin in H. lightfooti is indolelactic acid-spermine (Tipton & Dajus 1994). Symptoms of envenomation are vomiting, shock, and immobility (Bowles & Swaby 2006).

Despite its enormous size and grotesque appearance, the mygalomorph spiders’ venom is much less potent than that of the much smaller button spiders (Dippenaar-Schoeman 2002). There are two main species of button spider in southern Africa, Latrodectus geometricus (Brown button spider) and Latrodectus cinctus (Black button spider), both related to the infamous Black widow spider of North America. Latrodectus species are the most dangerous spiders to humans and can be fatal (Bowles & Swaby 2006). The venom of L. geometricus, the more venomous of the two southern African species, consists of complex proteins such as α-latrotoxin, a powerful neurotoxin (Reyes-Lugo et al. 2009). Symptoms typically include muscle pain, abdominal cramps, difficulty breathing, nausea, hypertension and localised necrosis around the bite area (Bowles & Swaby 2006; Reyes-Lugo et al. 2009). Bites may result in a chronic condition known as lactroderctism, which causes severe and persistent pain (Bowles & Swaby 2006). The proteins in Latrodectus venom have high molecular masses ranging from 43 000 to 220 000 (Petrenko et al. 1993).

Carabidae (ground beetles)
Although some of the best known San arrow poisons are made from Diamphidia or Polyclada beetle larvae, the carnivorous Lebistina beetles, including L. subcruciata, L. holubi and L. peringueyi, that are parasitic to these taxa, were also used in the same context (Shaw et al. 1963; Neuwinger 1996, 1998; Nadler 2005). It has been proposed that some southern African Lebistina species exhibit Batesian mimicry of the poisonous Diamphidia and Polyclada (e.g. Brandmayr et al. 2009). The toxicology and biochemistry of these animals, however, seem under-researched and have not been independently described from that of their hosts (see below). It is therefore not clear whether they display similar toxicity. The Lebistina parasite infests less than 5 % of cocoons and is not favoured by all tribes (Chaboo pers. comm. 2014).

Chrysomelidae (leaf beetles)
Several species of the Chrysomelidae family of leaf beetles are reported to have been used to poison arrows in the Kalahari. The main ones are Diamphidia simplex and Diamphidia nigro-ornata, found on Commiphora (myrrh) trees; Polyclada flexuosa, found on Sclerocarya birrea (marula) trees; and Blepharida vittata and Blepharida lewini. Although the adult beetles were sometimes used, usually the larvae, and sometimes the pupae, were harvested and their entrails squeezed directly onto the arrow shaft behind the arrow point (Schapera 1925; Silberbauer 1965; Lee 1979; Marshall-Thomas 2006). Although Diamphidia and Polyclada poison is only reported to have been used among hunter-gatherer communities living in the Kalahari, the Commiphora and Sclerocarya trees have
a much wider distribution. The *Commiphora* tree and the *Diamphidia* beetles have been recorded as far south and east as KwaZulu-Natal (Chaboo et al. 2007; Chaboo pers. comm. 2014).

For how long the diamphotoxin remains active is unknown. Twentieth-century San groups prefer to make a new batch every 6–12 months, after which the poison is thought to be useless (Marshall-Thomas 1959 cited in Robbins et al. 2012; Campbell & Lamont 1968). Pharmacological studies conducted on museum collections, however, show that some arrows thought to have been poisoned using the *Diamphidia* grub were still active after 80 years (Lewin 1894). Although, as noted below, this may be due to the presence of a longer-lasting plant alkaloid. Hall and Whitehead (1927) suggest that toxins released by micro-organisms such as bacteria may be responsible for the prolonged virulence of museum arrow specimens, rather than any longevity of intentionally applied poisons.

Whereas the Watt and Breyer-Brandwijk (1962) experiments indicated the rapid effect of *Diamphidia*-based poison, Silberbauer (1965) observed that on large game hunted by the San, such as wildebeest, kudu, oryx or eland, it can take up to 12–15 hours before the animals succumb. Neuwinger’s (1996) more recent observations support those of Silberbauer, indicating it to be a rather slow-acting but fatal poison during actual bow hunting, with death or immobility taking at least a few hours to occur. More often, the weakened animals are killed with spears several days after having been shot with poisoned arrows (Silberbauer 1965). Silberbauer (1965) notes that the larvae poison was seen as an emergency solution when more lethal and fast-acting plant-based poisons were not available. And, as an interesting aside, Neuwinger (1996: 893) observed people falling into a trance after having smoked a powdered grub mixed with tobacco.

Some studies have been undertaken to discover the source of the *Diamphidia* toxin. The main toxin, a toxalbumin originally noted by Lewin (1894), has been identified as a single-chain polypeptide protein called diamphotoxin, and has a molecular mass of 60 000 (De la Harpe & Dowdle 1980; De la Harpe et al. 1983). There appears, however, to be some confusion over whether it is the protein itself or another low weight (± 700) labile substance, possibly an amino acid, which binds to the protein that is responsible for the toxicity (cf. Mebs et al. 1982; Woollard et al. 1984; Kao et al. 1989). The study by Mebs and colleagues (1982) was the only one to identify the presence of a lighter weight substance. Haemolytic and neurotoxic effects have been attributed to the diamphotoxin, which is said to block neuro-muscular function (Campbell & Lamont 1963; De la Harpe et al. 1983; Mebs et al. 1982; Woollard et al. 1984; Kao et al. 1989). Death is thought to be due to haemolysis, tissue hypoxia and reduced oxygen carrying capacity (Kündig 1978; Mebs et al. 1982; Jacobson et al. 1989; Kao et al. 1989). The lethal dose is 5–20 μm/kg (Woollard et al. 1984). Extracts of *P. flexuosa* pupae are also strongly haemolytic and lethal to mice, but the active principle is different, and they did not display the dominant molecular mass of 60 000 found in *D. nigro-ornata* extracts (De la Harpe et al. 1983).

*Parabuthus* spp.

Scorpion venom is reported to have been used among hunter-gatherers from Lesotho (Schapera 1925), and the Kalahari (Nadler 2005). Nadler (2005) states that this ingredient is considered a ‘welcome additive’ to arrow poisons by poison makers. Although the method of preparation was not recorded for the San, scorpion venom
was also used as an ingredient of arrow poison among indigenous Americans, where it was dried and powdered (Bisset 1989). Unlike snake venom, the toxicity of scorpion venom is not affected by climate or geography (Debont et al. 1998). Scorpion venom is thought to function by inhibiting potassium channels and altering sodium channels in cells (Debont et al. 1998; Inceoglu et al. 2003). Potassium channelling is important for regulating and controlling neurons, with disruption resulting in hyper-excitability of the peripheral nervous system in vertebrates (Debont et al. 1998). The effects of scorpion envenomation are paraesthesia, muscle pains and cramps, difficulty breathing and trembling (Bisset 1989; Müller 1993).

Scorpion venom is a complex aqueous mixture containing mucus, inorganic salts, low molecular weight molecules, peptides and small proteins (Debont et al. 1998; Inceoglu et al. 2003). Two types of polypeptide chains have been discovered, a shorter one of 31–40 amino acids and a longer one of 60–70 amino acids joined by 3–4 disulphide bonds (Müller 1993; Debont et al. 1998). The latter have an estimated molecular weight of between 6000 and 8000 (Bisset 1989). In a recent study of Parabuthus transvaalicus venom, Inceoglu and colleagues (2005) identified three novel peptide toxins resembling the longer chain group, but consisting of 58 amino acids. These toxins are bestoxin, dortoxin and altitoxin, the latter two being lethal (Inceoglu et al. 2005). In addition, two smaller peptide chain toxins were identified, namely ilkitoxin and birtoxin, the latter being lethal (Debont et al. 1998; Inceoglu et al. 2005).

**PLANT POISON INGREDIENTS**

**Acacia** spp.

The *Acacia* tree is well known in the African savannah and bushveld biomes (Van Wyk & Van Wyk 1997). Many trees contain cyanogenic glycosides, saponins, tannins, alkaloids of simple structure, and derivatives of L-cystein and pippecolinic acid (see Neuwinger 1996). It is used as an additive to hunting poisons in various parts of the continent. In southern Africa the *A. Mellifera* Mill. and another unidentified species have been observed as additive ingredients in the *Diamphidia* poisons of the Ju’wasi (formerly !Kung) in northern Botswana and the Kalahari (Neuwinger 1996). It is not known why the Ju’wasi use *Acacia* sap in their poisons as it is not considered particularly virulent; nor does *Acacia* gum have any adhesive properties (Neuwinger 1996). Certain *Acacia* gums are known to be used as food stabilisers (Siew & Williams 2008) and could perhaps have been used for the same purpose in poison recipes.

**Aokakanthera** spp. (poison bush)

*Aokakanthera* is used as an ingredient of arrow poisons throughout Africa, either by itself or mixed with other ingredients (Watt & Breyer-Brandwijk 1962; Neuwinger 1996) and is probably one of the most well-known African plant poisons. Among the San of southern Africa the wood and leaves of *A. Oppositifolia* (Lamarck) Codd. were pounded and boiled until a gelatinous consistency was obtained (Von Wielligh 1921). This was then smeared directly onto the arrow or mixed with euphorbia latex (Smith 1888; Schapera 1925; Shaw et al. 1963; Bisset 1989; Neuwinger 1996). *A. laevigata* (Lebzelter 1996 (1934, listed as *A. venenata*); Neuwinger 1996), *A. schimperi* (Schwein f.) (Neuwinger 1996) and *A. spectabilis* (Nadler 2005) have also been recorded as arrow poison ingredients.
The sap of the *Acokanthera* plant contains the lethal cardiac glycoside acovenoside A (Cheeke 1989; Kumar & Singh 2005), whose effect reputedly is similar to that of ouabain (Shaw et al. 1963; Kumar & Singh 2005). Symptoms of *Acokanthera* poisoning include involuntary twitching, respiratory distress, irregular heartbeat, elevated blood pressure and finally cardiac arrest (Neuwinger 1996). Intravenous contact may result in death within 20 minutes (Kumar & Singh 2005). In addition to acovenoside A, the plant also contains trypsin and chymotrypsin proteinase inhibitors (Kumar & Singh 2005), and is regarded as dangerous to animals (Curson 1928). The organic chemistry of *Acokanthera oppositifolia* is well studied and a complete list of toxic compounds may be found in Neuwinger (1996).

**Adenium spp. (desert rose)**

Several species of *Adenium* are used for arrow poisons throughout Africa, including *A. Boehmianum* (Schinz), *A. coetaneum*, *A. swazicum* and *A. honghel* (Watt & Breyer-Brandwijk 1962; Bisset 1989; Neuwinger 1996; Nadler 2005). The most common, however, is *A. Obesum* (Forskal) Roemer et Schultes, widely used in East Africa (Neuwinger 1996, 2004). The latex or the concentrated root sap is often directly applied to the arrows (Neuwinger 1996). Neuwinger (1996) reports that *A. boehmianum* poison used by Hei||om bushmen and the Damara of Namibia causes death within an hour or less for springbok, between 2–3 hours for kudu, oryx, hartebeest and wildebeest, and between 4–5 hours for eland.

The most common southern African species, *A. boehmianum* and *A. Multiflorum* (Klotzsch), contain several cardiac glycosides present in the sap. These include: obebioside B, odorotrioside G, tetraphyllin B, hongheloside A-F, somaline and echujine, among others (Cheeke 1989; Neuwinger 1996; Van Wyk et al. 2002). Adenium poison can be used alone or in combination with other plant poisons, usually euphorbia or *Spirostachys africana* (Neuwinger 1996). Symptoms in cats include incontinence, restlessness, rapid breathing and convulsions followed by death within 20 minutes of intravenous administration (Neuwinger 1996).

**Bobgunnia madagascariensis** (also *Swartzia madagascariensis*) (snake bean)

The seed pods of this species have been used since ancient times in Africa. Fagan and Van Noten (1971) report the presence of *Swartzia* seed pods in archaeological excavations in south Zambia, where they are interpreted as possible poison ingredients. In 1981 Neuwinger observed the use of this plant during every poison-making event in the Okavango-Caprivi region, as well as in much of East Africa (Neuwinger 1996). As recently as 2000, Nadler (2005) observed how the legume flesh of the plant was used as an arrow poison ingredient in Namibia. He describes how after the ‘snake bean’ is roasted over an open fire, its flesh is then removed and grated into small fragments, before being mixed with other ingredients.

The stem-bark and fruit of this plant are also used as fishing poisons (Neuwinger 2004). Despite the plant’s wide ranging usage in hunting and fishing poison recipes, Neuwinger (1996) does not consider it to be particularly toxic. Mice that received *B. madagascariensis* Kirkbr et Wiersema exhibited signs of buccal and nasal irritation with occasional sneezes followed by a lethargic stance prior to death (Nyahangare et al.
The fruit contains catechin-tannins, a strongly haemolytic saponin, a mixture of oleanolic acid and O-acetyloleanolic acid, gypsogenin and kaempferol (Jewers et al. 1971; Neuwinger 1996; Hostettmann & Marston 2002).

*Boophane disticha* (poison bulb)

*B. disticha* (Linne f.) Herbert is probably one of the most important plants in southern Africa, not only for its prominent role in arrow poisons (e.g. Dornan 1925; Lebzelter 1996; Nadler 2005), but also for its medicinal and recreational properties (Neuwinger 1996; Van Wyk & Gerike 2000; Du Plooy et al. 2001; Van Wyk et al. 2002). The use of *B. disticha* in southern Africa stretches back many millennia. For example, Johan Binneman (1999) identified *Boophane* scales wrapped around a 2000-year-old mummy from an archaeological site in the Eastern Cape (Binneman 1999), and a 6000-year-old stone tool from Melkhoutboom, in the same province, was also found wrapped in the remains of this plant (Deacon & Deacon 1999).

*B. disticha* poison was apparently intended to disorientate the quarry, while more potent ingredients were used to kill it (Kannemeyer 1890 in Deacon & Deacon 1999). Numerous toxic alkaloids have been identified in the *B. disticha* bulb, including buphandrine, buphanine, distichamine, lycorine, crinamidine, haemanthin and eugenol, a volatile oil with analgesic properties (Lewin 1912; Shaw et al. 1963; Cooke & Warren 1953; Neuwinger 1996; Du Plooy et al. 2001; Van Wyk et al. 2002). A number of glycosides are also present but these do not appear to have any toxic effects (Neuwinger 1996). A concoction of the bulb taken orally causes sedation, analgesia, visual hallucinations, irrational behaviour and, occasionally, coma and death (Watt & Breyer-Brandwijk 1962; Du Plooy et al. 2001; Van Wyk et al. 2002). Lewin (1912) described the alkaloid haemanthin as exhibiting narcotic properties similar to atropine.

**Cucurbitaceae (melons/cucumbers)**

Nadler (2005) has recorded the use of several cucumber species by the Ju|wasi as ingredients in arrow poisons. For example, in southeast Angola he observed the use of the taproots of *Acanthosicyos naudiniana* (Sonder) C. Jeffrey (*Citrillus naudinianus*; Nadler 2005). Small pieces of the chopped root are boiled for several hours in water until a thick plant extract is obtained, which is then used to poison arrows (Neuwinger 1998; Nadler 2005). Dorothea Bleek (1928) describes how *Citrillus lanatus* (Thunb.) roots are warmed in hot ashes, beaten against the ground and wrung out to obtain the juice for mixing arrow poison. Among the hunter-gatherers of Namibia and Botswana, particularly the Ju|wasi, Kxoe and !Ko, the root sap is used as an additive to the *Diamphidia* poison (Bleek 1928; Neuwinger 1996; Nadler 2005).

*A. naudiniana* belongs to the Cucurbitaceae with the highest level of toxicity. It contains various cucurbitacins with the highest concentration found in the roots of the plant (Nadler 2005). Cucurbitacin is also the toxin found in the other cucumber species. Cucurbitacins manifest as glycosides and are generally cytotoxic (Alghasham 2013). In an experiment where rabbits ingested cucurbitacin-containing bitter melons (Steyn 1950 in Neuwinger 1996), as little as 2 g per kg body weight, either fresh or cooked, proved fatal. The poison mainly affects respiration and the gastro-intestinal tract, with some symptoms resembling strychnine poisoning (Neuwinger 1996).
Euphorbiaceae (spurge family)

Members of the Euphorbiaceae family are probably the most widely and commonly used ingredients in arrow poisons, both in southern Africa (see Shaw et al. 1963) and the rest of the continent (Bisset 1989; Neuwinger 1996). The family comprises many species, including the castor bean plant from which ricin is derived. In this section, however, we deal only with the cactus-like, succulent *Euphorbia* species. The three species most commonly reported to be used as hunting poisons in southern Africa are the *E. ingens* (E.Mey ex Boiss), *E. virosa* Willdenow and *E. arborescens* (see Shaw et al. 1963), of which *E. virosa* is considered to be the most virulent (Watt & Breyer-Brandwijk 1962; Kontiswe 2013). To this list may be added *E. Tirucalli* (Linne) and *E. coerulescens*, both of which contain potent diterpenoids (Morton 1958; Evans 1978; Neuwinger 1996; Van Wyk et al. 2002). Euphorbias are also the most widely used fishing poison recorded (Neuwinger 1996, 2004).

*Euphorbia* latex causes inflammation, skin irritation, conjunctivitis of the eyes, and burning of the oral cavity and throat (Neuwinger 1996). The carcinogenic latex contains various serine proteases (Konno 2011), terpenoids such as euphol and phorbol, lectins, and several esters of diterpene alcohols (Opferkuch & Hecker 1974; Neuwinger 1996; Konno 2011). Six irritant esters of low toxicity have been isolated from *E. coerulescens*, including 12-deoxyphorbol, trimesters of phorbol and three monoesters of the same diterpene (Evans 1978). Full pharmacological and chemical analyses of *Euphorbiaceae* and many other poisonous plants in Africa can be found in Neuwinger (1996). Besides being used as poisons, euphorbias have many medicinal uses due to the anti-microbial properties of the latex (Ramavhoya 2005; Langat et al. 2012).

*Hyaenanche globosa* (hyena poison)

*H. globosa* (Lamb.) is endemic to the Varnhynsdorp area of the Western Cape (Van Wyk et al. 2002). The powdered seeds were used as an ingredient in San arrow poison (Schapera 1925; Watt & Breyer-Brandwijk 1962). Today the seed of *H. globosa* is still commonly used for treating carcasses to kill hyenas, jackals and similar predators. The fruit is pounded into a powder and administered in the same manner as *Nux vomica* (Lambert 1797).

Henkel (1913 cited in Watt & Breyer-Brandwijk 1962) first isolated a toxic principle from the seed. This was a sesquiterpenoid lactone called hyaenanchin and now known as tutin (Van Wyk et al. 2002). Tutin poisoning causes severe convulsions, leading eventually to death (Zhou et al. 2006). The fruit and seeds have the most highly concentrated portion of this toxin (Watt & Breyer-Brandwijk 1962). Tutin is water soluble and therefore a formidable poison (Watt & Breyer-Brandwijk 1962). Although considered a lethal poison, there are no recent reports of human or livestock poisoning (Van Wyk et al. 2002).

*Pachypodium* spp. (bottle tree)

*P. lealii* is listed by Shaw and colleagues (1963; also see Watt & Breyer-Brandwijk 1962) as an arrow poison ingredient of the Bergdama. The species is endemic to Namibia and southern Angola. The sap from the plant contains several cardiac glycosides (Nadler 2005; Bester 2007). *Pachypodium* is closely related to *Adenium*, and falls in a group of the Apocynaceae family that is notorious for yielding potent poisons.
P. lealii contains the cardiac glycoside pachypodiin (Watt & Breyer-Brandwijk 1962; Cheeke 1989).

**Protasparagus exuvialis**
The sap of the pod-like roots of this plant is listed as an additive ingredient in the *Diamphidia* arrow poison of the Ju'wasi (Neuwinger 1996; Nadler 2005). Neuwinger (1996) noted that a poison maker would expect to develop a high fever coupled with debility and confusion, should any of the root sap get into a cut in the skin. The chemistry of this plant does not appear to have been studied.

**Sanseviera** spp.
*Sanseviera* species have a global distribution. *S. angolensis* was harvested for its fibrous properties and turned into rope for various purposes (Chapman 1864). Some are known for their antiseptic properties (Neuwinger 1996; Philip et al. 2011) and *S. aethiopica* (Thunb.) is reported to have been used by the Zhu and Ju’wasi San to strengthen and prolong the lifespan of *Diamphidia* poison (Neuwinger 1996; Nadler 2005; Robbins et al. 2012). All *Sanseviera* species thus far tested were toxic to mice, and *S. trifasciata* extracts were fatal (Neuwinger 1996). *S. aethiopica* does not seem to have been fully investigated for the presence of toxins, but is reported to contain haemolytic saponins (Neuwinger 1996). Preliminary phytochemical screening of the extracts from *S. trifasciata* showed the presence of alkaloids, flavonoids, saponins, glycosides, terpenoids, tannins, proteins and carbohydrates (Anbu et al. 2009).

**Solanum** spp.
*Solanum* species belong to the potato family and include the notorious European species known as deadly nightshade (*Atropa belladonna*). *S. kwabense* is another species previously listed by Neuwinger (1996), and recently observed as being used as an arrow poison ingredient by the Ju'wasi of Namibia (Nadler 2005). *S. kwabense* and *S. incanum* (Linne) may be used alone or as an additive to *Diamphidia* poison, where the grubs are available (Neuwinger 1996).

The plant causes the so-called ‘maldronksiekte’ (crazy-drunk illness) in cattle, characterised by epileptic-like fits during which an animal staggers around with an extended and slightly twisted neck, and sometimes falls to the ground (Van der Lugt et al. 2009). Severe haemorrhagic gastroenteritis is generally seen, and death is caused by cardiac or respiratory arrest (Neuwinger 1998). Symptoms include vomiting, diarrhoea, rapid pulse and breathing, cramps and paralysis (Van Wyk et al. 2002).

Most *Solanum* species produce poisonous steroidal alkaloids of saponin character such as the glycoalkaloids solasonine and solamargine (Neuwinger 1996). The fruit contains the highly toxic N-dimethylnitrosamine. A number of glycoalkaloids found in the plant have been shown to produce cardiotonic effects similar to those of cardiac glycosides (Neuwinger 1996, 1998).

**Spirostachys africana** (tamboti)
Neuwinger (1996) reports the recent use of tamboti as an arrow poison used by the Ju’wasi at Grootfontein in Namibia, and describes the current role of the tree in fish poisoning in southern Africa (Neuwinger 2004). The sap from this tree was used as
an additive ingredient among the Hei|om tribes of Namibia, together with *Euphorbia* latex and *Adenium* juice (Fourie 1926). The stem bark also has antibacterial properties and is used traditionally for the treatment of dysentery (Mathabe et al. 2008).

*S. africana* (Sonder) sap contains several diterpenes, including stachenone and stachenol (Van Wyk et al. 2002), as well as two triterpenoids, including 3-acetyl aeritoxic acid and lupeol (Mathabe et al. 2008). In addition, Munkombwe and colleagues (1997, 1998) isolated various beyerene diosphenols and a nor-keto acid (also see Neuwinger 1996).

**Strophanthus spp.**

Species of this plant are the most widely used plant poisons in Africa and occur as a base ingredient in many poison recipes throughout the continent (Bisset 1989; Neuwinger 1996, 1998). Two species, namely the *S. kombe* (Oliver) and *S. speciosus* occur in eastern and southern Africa, from south-eastern Kenya and eastern Tanzania to eastern Namibia (Caprivi Strip), Botswana, Zimbabwe, Mozambique and northern South Africa. Ju|wasi informants described how crushed seeds were used in hunting poisons (Dornan 1925; Nadler 2005). The same use and method of preparation is practised in Kwazulu-Natal by the Zulu (Watt & Breyer-Brandwijk 1962). Using a goat, it was demonstrated that death occurs within a few seconds after administering *S. kombe* (De Villiers 1949 in Neuwinger 1996). *Strophanthus* is also reported as a fishing poison (Neuwinger 2004) and a hallucinogenic (Sobiecki 2002).

More than a dozen cardiac glycosides (cardenolides) have been isolated from *S. kombe* and these are present in high concentrations in the roots and seeds (see Neuwinger 1996). The major component in *Strophanthus kombe* is k-strophanthoside. Other glycosides in the mixture, based on the aglycone strophanthidin, are cymarin, g-strophanthin-β (also known as ouabain), erysimoside and helveticoside, and several minor components have also been isolated (e.g. Hollman 1985; Cheeke 1989). All compounds are highly toxic. The glycosides are most abundant in the seeds of the plant, with about 4% of the seed comprising toxic glycosides (e.g. Philipe & Angenot 2005).

**Strychnos spp. (strychnine)**

Because of their toxicity, many *Strychnos* species have been used throughout the world as arrow poisons or in ordeals (Bisset 1989; Philipe & Angenot 2005). The use of certain species to induce trance has also been reported (Sobiecki 2002). Although the famed species *S. nux-vomica* and *S. toxifer* are not found in Africa, there are several species that are indigenous to Africa and southern Africa, such as the *S. madagascariensis* (Poir.), *S. spinosa* (Lamarck) and *S. Usambarensis* (Gilg). Strychnos species have been reported as a substitute for snake venom in arrow poisons among certain Hei|om tribes in Namibia (see Hall & Whitehead 1927). Dornan (1925) lists *Strychnos toxifer* as a San arrow poison ingredient, but this was most likely a misidentification (see Shaw et al. 1963).

Not all *Strychnos* species indigenous to southern Africa are very poisonous, but the local *S. usambarensis* contains more than 60 alkaloids (see Neuwinger 1996 for a detailed list). Strychnine is not present in *S. usambarensis*, but several curare-like alkaloids make it an effective fish poison (Neuwinger 1996; Philipe et al. 2004). The *S. madagascariensis* fruit is eaten in South Africa, but trace amounts of strychnine and C-toxiferine have been reported from the unripe seeds (Mawela 2008). Various saponins, sterols and
terpenes have been reported in *S. spinosa* (Neuwinger 1996). This plant is widely used as an arrow poison in Central Africa (Neuwinger 1996) but has not been recorded in this context in southern Africa, despite its abundance in the eastern portion of the subcontinent.

It is likely that geographic factors affect the presence and potency of toxic compounds, as an *S. spinosa* tree grown in Florida, USA, tested negative for all toxic compounds (Lofgren & Kinsley 1942). Alkaloids previously only known in South American calabash curare and *Styphnolobium* species were obtained from *S. usambarensis*, and the poisons derived from its roots include: C-dihydrotoxiferine, C-curarine, C-calchassine and afrocurarine (Neuwinger 1996, 1998). Afrocurarine causes neuromuscular blocking resulting in complete skeletal and muscular paralysis (Neuwinger 1998). The usumbarine-type alkaloid isostrychnopentamine, the main ingredient of African curare arrow poison, has also been identified (Philipe & Angenot 2005).

*Terminalia sericea* (Burch.) (silver cluster-leaf)
Nadler (2005) observed the Ju|wasi using both the branches and leaves in their poisons, whereas it was listed by Silberbauer (1981) only as an adhesive. Neuwinger (1996) also lists it as an additive in *Diamphidia* poison recipes among the [Gwi and Ju|wasi on the Namibian/Botswana border. *Terminalia* species are considerably toxic. For example, Neuwinger (1996) reports a case in Tanzania in which *T. sericea* caused severe abdominal pains, vomiting, purging and frequent urination, followed by death after 19 hours. Sericic acid and its glucoside sericoside were isolated from the roots, as well as the glycoside resveratrol 3-O-rutinoside. *Terminalia* species are generally rich in acid triterpene saponins and tannins, but little else seems known about the toxins associated with the genus (Neuwinger 1996).

*Urginea* spp.
Dornan (1925) listed *Digitalis purpurea* as an arrow poison ingredient, but Nadler (2005) explains that the correct species is most probably *U. sanguinea* (Schinz) (also known as *Drimia sanguinea*, or slangkop (snake head)), which is very similar to the European *D. purpurea* with which Dornan would have been more familiar. Kholer (1973 in Neuwinger 1996) also listed the bulb sap of *U. sanguinea* as an arrow poison ingredient of Kxoe hunter-gatherers of Namibia. *U. epigea* (Dyer) has been mentioned in the context of arrow poisons by Kholer (1973, cited in Neuwinger 1996) and Bisset (1989) among the Kxoe in the Caprivi Strip. All the parts of *Urginea* are highly toxic (Neuwinger 1996), and acute cardiac glycoside poisoning frequently occurs in livestock that have ingested it (Van Wyk et al. 2002; Botha & Penrith 2008). Symptoms typical of cardiac glycoside poisoning include respiratory distress, severe diarrhoea and weakening of the hind quarters (Neuwinger 1996). *U. epigea* is one of the main culprits behind stock losses in southern Africa (Curson 1928; Kellerman et al. 1988; Van Wyk et al. 2002). Scillaren A (Transvaalin) is the lethal cardiac glycoside isolated from *U. sanguinea* (Neuwinger 1996). The *U. epigea* plant contains several cardiac glycosides including scillaren A, proscillaridin A, physodine A, urginin and 2α-epoxyscillirosidine (Cheeke 1989; Krenn et al. 1993; Van Wyk et al. 2002; Koorbanally et al. 2004). Some of these toxins are also found in *Drimia robusta* and *Homeria pallida* (Van Wyk et al. 2002).
BACTERIA

Klipgift (rock poison) and/or bacteria

Originally reported by Barrow (1806: 194) and described as a thick black substance with a bituminous odour growing on the roof of a rock shelter in the Sneeuberg, this substance is reputed to be a virulent arrow poison (see also Hahn 1870; Fritsch 1872; Lichtenstein 1930). The exact method of preparation, however, has never been recorded. Schapera (1925) thought it to be a form of arsenic, whereas Shaw and colleagues (1963) ascribed it to dassie (Procavia capensis) urine, also known as hyraceum, which is reported to have been used in some areas as a poison ingredient (Watt & Breyer-Brandwijk 1962). Hyraceum, although a virulent irritant if it gets into your eyes (Chase pers. comm. 2009), does not accumulate on shelter roofs, nor is it thought to be particularly poisonous, and is used in perfumes and traditional medicine (Khoza & Hamer 2013).

It could be that we are rather dealing here with a type of cyanobacteria. Arsenic is a mineral and is present in rocks rather than as a film on their surface. On the other hand, more than 30 species of terrestrial cyanobacteria have been identified in South Africa, one of which (Chroococcidiopsis) produces a strong neurotoxin (Büdel 1999; Cox et al. 2005). Many other species produce neuro- and hepatotoxins (Cox et al. 2005). Cyanobacteria and cyanobacterial lichens grow on exposed rock surfaces under varying climatic conditions and are responsible for the black colour on many rock faces, such as those of the Golden Gate Highlands in the eastern Free State (Büdel 1999; Hoffman et al. 2003; Venter et al. 2010; Albertano 2012). We are not yet aware of any studies that have been conducted on cyanobacteria from the Sneeuberg or Cape region, but toxic species might be present there too.

Hall and Whitehead (1927) considered bacteria, of the soil flora variety, to be responsible for the longevity of some arrow poisons. They suggest that whereas plant and animal poisons would degrade over time, bacteria remain active for much longer, perhaps accounting for the longevity of some arrow poisons. Some hunter-gatherer tribes would dip their arrows into the putrefying carcass of an animal before they went hunting (Hall & Whitehead 1927). Their analysis of several Hei|om arrowheads tested positive for the presence of pathogenic bacteria, to which they attribute the efficacy of the arrow ‘poison’ (Hall & Whitehead 1927).

Appendix B

Other potential sources of hunting poisons

Apart from the plants and animals already mentioned, many other poisonous species exist in southern Africa that are more or less suitable to be used for poisoning arrowheads (see Neuwinger 1996; Van Wyk et al. 2002 for lists of poisonous plants). Two of the criteria that must be met are: 1) the toxic substance must be easily applicable to an arrowhead, and 2) the substance must be toxic intravenously without causing the meat to be poisonous to consumers. Many plants cause poisoning only if ingested; these would not be suitable as arrow poisons. Several southern African fungi species are also highly toxic and suitable for arrow poisons (Watt & Breyer-Brandwijk 1962).
What follows is information on poisonous plants and animals that have not yet been implicated in San hunter-gatherer arrow poisoning, but which nevertheless could have been used in this context.

ANIMAL SOURCES

*Phrynomerus bifasciatus* (red-banded rubber frog)

Few pharmacological studies have been conducted on the skin secretions of the red-banded rubber frog, which is a nocturnal frog occurring in northeastern South Africa, Mozambique, Zimbabwe and farther north, extending also into northern Namibia (Du Preez & Carruthers 2009; SA Reptiles 2010). In South Africa its distribution roughly parallels that of the *Commiphora africana* tree, on which the *Diamphidia* beetle feeds. The use of frog secretions as arrow poisons is well known among the hunter-gatherer tribes of South America but has not been documented in southern Africa. The toxin of the red-banded rubber frog is unidentified beyond the fact that it is cardiotoxic (Van der Walt et al. 1992).

Among the tropical frogs of South America, the toxic components of the skin secretions have been identified as alkaloids, probably derived from the frogs’ diet of ants (Daly et al. 1987). The red-banded rubber frog’s diet also consists of ants and termites (SA Reptiles 2010), one species of which, *Solenopsis punctaticeps* or the fire ant, contains the toxic alkaloid trans-2-n-butyl-5-n-pentylpyrrolidine (Jones et al. 1982). The skin secretion of the red-banded rubber frog is milky and very sticky (B. Muller pers. comm. 2014) and could conceivably be used as an arrow poison.

Although no formal studies have been done on the red-banded rubber frog toxins, we are fortunate to have two accounts of accidental exposure to the skin secretion (Jaeger 1971; Pickersgill 1997). Both authors describe similar symptoms. These include tingling around the area of contact, tachycardia, difficulty breathing, dizziness and nausea. Symptoms began approximately 30 minutes after contact and lasted about 4 hours (Jaeger 1971; Pickersgill 1997). It is conceivable that, had larger amounts of the secretion entered the bloodstream, the symptoms would have been amplified, thus making it an effective ingredient in an arrow poison.

PLANT SOURCES

*Alou* spp.

Most *Aloe* species can be used medicinally to treat various ailments (Van Wyk & Gericke 2000; Gaffney 2006; Mawela 2008; Schmelzer & Gurib-Fukkim 2008). Approximately 16% of African *Aloe* species contain toxic piperidine alkaloids and have been observed as hunting poisons in East Africa (Neuwinger 1996). There are two species of *Aloe* occurring in southern Africa that are considered to be highly poisonous, namely, *A. gariepensis* (Pillans) and *A. globuligemma*. Neither is reported to have been used in the context of hunting, however. These species contain the same toxic alkaloids found in poison hemlock (*Conium maculatum*), namely, coniine, conhydride and γ-coniceine (Neuwinger 1996; Reynolds 2005; Schmelzer & Gurib-Fukkim 2008). Poisoning by *A. globuligemma* has been reported to result in haemorrhagic lesions leading to death within 36 hours (Parry & Matambo 1992). *A. zebrina*, a species not known for its toxicity, was nevertheless used by the G|wi to bind the *Diamphidia* poison to their arrows (Robbins et al. 2012).
**Abrus precatorius** (Linne) (crab’s eye creeper)

Watt and Breyer-Brandwijk (1962) list *A. precatorius* as used in arrow poisons. Injected intravenously or subcutaneously, very small quantities of the seed extract are lethal. After injection, there is a latent period of several hours up to a day or two, before the symptoms appear. Symptoms include loss of appetite, vomiting and diarrhoea. Post-mortem observations include inflammation of the intestinal mucosa and haemorrhagic effusions into the body cavities and organs (Watt & Breyer-Brandwijk 1962).

The plant seed contains the toxin called abrin (A and C), which is very similar in structure and properties to ricin (Wei et al. 1974; Van Wyk et al. 2002; Dickers et al. 2003). Both abrin and ricin have the same molecular weight (Jung-Yaw et al. 1970) making differentiation by mass spectrometry virtually impossible. The symptoms of these toxins are severe stomach pains diarrhoea, nausea, cold sweat and drowsiness, followed by intestinal inflammation and haemorrhage (Van Wyk et al. 2002). Abrin C was found to be more toxic on mice than abrin A (Wei et al. 1974).

**Afzelia** spp.

The roots of the *A. quanzensis* (Welw.) and *A. africana* (Sm.) are used medicinally in Botswana as a remedy for bilharzia, but the roots are used as a hunting poison in Tanzania and Uganda (Neuwinger 1996).

**Amaryllis belladonna** (Linne)

The *A. belladonna* is endemic to the Western Cape Province and is responsible for several stock losses (Van Wyk et al. 2002). The bulb and seeds are very poisonous, with 200 g being sufficient to kill a sheep (Van Wyk et al. 2002). The bulb contains several isoquinoline alkaloids, of which the major one, ambelline, is said to have an analgesic effect similar to that of morphine (Van Wyk et al. 2002). Other toxins identified include lycorine, acetylcaranine, caranine, amaryllidine and undulatine (Watt & Breyer-Brandwijk 1962; Van Wyk et al. 2002). Intramuscular injection of the alkaloidal mixture in dogs produces muscular stiffness and incoordination, and stimulation of the respiration with subsequent depression of it. Intramuscular injection causes vomiting and diarrhoea, with death following within four hours from generalised collapse due to paralysis of the central nervous system (Watt & Breyer-Brandwijk 1962).

**Ammocharis coranica**

Part of the same family as *Boophane disticha* and *Amaryllis belladonna*, the *A. coranica* shares many of their toxic alkaloids. These include lycorine, caranine, crinamine and acetylcaranine, among others (Watt & Breyer-Brandwijk 1962; Munday 1988; Koorbanally et al. 2000).

**Annona chrysophylla** (Pers.)

Members of the Annona genus are used throughout Africa as additive ingredients in hunting poisons, and in South Africa the Venda add the bark of *A. chrysophylla*, a skin irritant, to their *Acokanthera oppositifolia* arrow poisons (Neuwinger 1996). Not much is known of the chemistry of this plant, but several diterpenes and isoquinoline alkaloids, including liriodenine, isoboldine and anonaiine, have been identified (see Neuwinger
1996). Several cyanogenic glycosides have also been reported but not characterised (Neuwinger 1996). *A. chrysophylla* occurs widely in the eastern half of southern Africa.

*Asparagus africanus* (Lamarck)

*A. africanus* is widely distributed in Africa. It has been recorded as an additive in the *Strophanthus* poisons of the Fula and Soce tribes of Nigeria and Senegal, and is used as a medicinal tea in Namibia and Zimbabwe (Neuwinger 1996).

*Brunsvigia radulosa* (Herb.)

This plant, found throughout southern Africa, is considered poisonous due to the presence of lycorine and crinamine (Watt & Breyer-Brandwijk 1962; Munday 1988).

*Cassia singueana* (Schinz)

The bark and roots are used as additives to *Acokanthera* poison in Kenya and as a cure for gynaecological problems among the Shona in Zimbabwe (Neuwinger 1996). The roots are reported to contain chrysophanic acid and torosachrysonne, but no alkaloids (Neuwinger 1996).

*Combretum* spp. (bush willow)

This species is found in the tropics. Certain tribes regard the root and fruit of the *C. erythrophyllum* as poisonous but give small doses of the root as a fattening tonic and to treat venereal diseases (Watt & Breyer-Brandwijk 1962; Le Roux 2003).

*Conium* spp.

*Conium* sp. could be responsible for the residues found on the Sibudu arrow tips (Matheson 2013). Several *Conium* species are indigenous to southern Africa, namely, *C. chaerophylloides*, *C. fontanum* and *C. sphaerocarpum*, although there is some confusion about the origin of *C. chaerophylloides* (cf. Gonçales 1978; Hilliard & Burtt 1985). Eight toxic piperidine alkaloids have been identified in *C. maculatum*, the most toxic of which are coniine and γ-coniceine (Lopez et al. 1999; Zabolotnyi et al. 2012). Although not much is known about the phytochemistry of South African *Conium* species, *C. chaerophylloides* has been shown to contain the same toxic alkaloids as the European variety 2-propyl-5-hydroxy-N-methylpiperidine (5-hydroxy N-methylpseudoconhydrine), a 5-hydroxy substituted piperidine compound (Roberts & Brown 1981). Zabolotnyi and colleagues (2012) have calculated the infra-red (IR) spectra for each of the conium alkaloids, so we are now able to identify their presence easily and non-destructively.

Conium alkaloids are neurotoxins (Reynolds 2005) and may be transferred to milk and fowl muscle tissue (Lopez et al. 1999). As a result, the meat of fowls poisoned with conium and the milk of animals that have ingested conium are not suitable for human consumption. We are not aware of any studies that have investigated whether mammal meat is edible after poisoning. Poison symptoms in cattle that have ingested the plant include muscle weakness, disorientation, trembling, cold limbs and excessive salivation. This is usually followed by overstimulation of the central nervous system, shallow respiration and dilated pupils, leading to coma and death (Lopez et al. 1999). Death occurs when the phrenic nerve in the medulla is excited, causing paralysis of the respiratory muscles (Lopez et al. 1999).
**Hoodia parviflora** (Sweet ex Decne)

The Hoodia plant is perhaps most well known for its role in dietary weight loss supplements (Van Wyk & Gericke 2000; Lynch et al. 2013). The plant contains abundant saponins and glycosides and is purported to have been used by the Himba in Kaokoland in bait to hunt jackal and small animals (Neuwinger 1996).

**Erythrophleum guineense** (Afzel ex R.Br.) (forest ordeal tree)

*Erythrophleum guineense*, known as the ordeal tree or red-water tree, is found from the Cape to East Africa. The bark, which is an article of commerce, is highly poisonous. The bark has been used by some Bantu-speaking tribes as an arrow poison, a fish poison, a poison for trial by ordeal, for medicinal purposes and as a tanning material (Watt & Breyer-Brandwijk 1962; Okeyo 2006).

Symptoms of intoxicification in humans include a reduced heart rate, followed by acceleration, dyspnoea with laboured respiration and death by respiratory arrest (Watt & Breyer-Brandwijk 1962). Death is due to cardiac and respiratory paralysis. The alkaloids, which include cassaidine, cassaine, cassamine, homophleine, erythrophleine and erythroplamine, also induce intense and long-lasting local anaesthesia, accompanied in most cases by irritation of the tissues concerned (Watt & Breyer-Brandwijk 1962).

**Fockea multiflora** (K. Schum.)

The plant contains an abundance of milk sap containing saponins and glycosides (Neuwinger 1996). The Himba of northern Namibia use the sap to poison their arrows as well as to poison carcasses in order to kill predators (Watt & Breyer-Brandwijk 1962; Neuwinger 1996).

**Kigelia africana** (Lam.) Benth.

*K. africana* is not known as a poison in southern Africa, but this use has been recorded in Central Africa, where it is used as an additive to *Strophanthus* and *Strychnos* poisons (Neuwinger 1996). In South Africa the Venda use the plant for medicinal purposes (Neuwinger 1996). Alkaloids present in the seeds and presumed to be responsible for their toxicity have not been identified (Neuwinger 1996).

**Millettia grandis** (E.Mey.) Skeels

*M. grandis* is found widely in the Eastern Cape and KwaZulu-Natal provinces. The bean is poisonous if ingested in quantity (Watt & Breyer-Brandwijk 1962). The powdered root can be used as a fish poison and the ground seed is suitable for arrow poison (see Watt & Breyer-Brandwijk 1962; Baloyi & Reynolds 2004).

The roots of the tree have apparently been used as arrow poison (Watt & Breyer-Brandwijk 1962; Van Wyk & Gericke 2000). The seeds are poisonous when eaten in large quantities, but when they are ground up and soaked in milk they provide a remedy for roundworm. The powdered roots can also be used to induce sleep and as a tranquiliser.

**Mundia whitei** (Hook.f.) Skeels

This plant is believed to have been used as additive in *Strophanthus* arrow poisons in Central Africa (Neuwinger 1996; Hutchings 1996 in Van Wyk & Gericke 2000). The shrub is found in southern Africa where it is used medicinally by the Zulu and
Shona tribes (Neuwinger 1996). While the phytochemistry of the shrub has not been extensively studied, the roots of the plant showed a strong presence of saponins, phenols, tannins and alkaloids with anthranal/phenolic glycosides (Neuwinger 1996; Gakunga et al. 2013). The seeds are considered to have been used as arrow poisons by some (e.g. Bester 2009).

*Mundulea sericea* (Willd.) A. Chev.
The pounded bark, leaves and sometimes seeds and roots are used for fish poisoning in Botswana and Zimbabwe (Watt & Breyer-Brandwijk 1962). Leaves are stripped and pounded, placed in bags and thrown into the water. The dead fish are then collected for food at considerable risk, since there are cases where people have become ill after eating the poisoned fish, although the leaves and bark are eaten by cattle and elephants with no ill effects (Watt & Breyer-Brandwijk 1962; Van Wyk et al. 2002; Bester & Grobler 2008). *M. sericea*’s role in arrow poisons is not well established but reports do exist (see Van Wyk et al. 2002). *M. sericea* contains potent rotenoids including rotenone, deguelin and tephrosin (Van Wyk et al. 2002). An unnamed glycoside has also been reported (Watt & Breyer-Brandwijk 1962).

*Ornithogalum* spp.
As many as fifteen species of *Ornithogalum* are found in southern Africa, all of which are lethally poisonous and have caused stock losses (Curson 1928; Watt & Breyer-Brandwijk 1962; Botha et al. 2000). *Ornithogalum* species are found primarily in the Cape ecozone. The bulb is thought to contain the highest concentration of toxins. Symptoms of ingestion include diarrhoea, fever, tachycardia, and severe abdominal pain, followed by convulsions leading to death (Watt & Breyer-Brandwyk 1962; Botha et al. 2000). Eight dried and powdered flowers are enough to kill a horse within 24 hours (Watt & Breyer-Brandwijk 1962). Chemical analysis of *O. thyrosoides* Jacq. revealed the steroid glycoside, prasinoside G, to be the primary toxin (Botha et al. 2000; Van Wyk et al. 2002), but other species have tested positive for cardiac glycosides and haemolytic saponins, including ipuranol and colchicine (Watt & Breyer-Brandwijk 1962; Van Wyk et al. 2002).

*Nicotiana* spp. (wild tobacco)
Various species of wild tobacco are found throughout Africa, some of which, such as *N. tabacum* and *N. rustica*, have been implicated as additives in the *Strophanthus* arrow poisons of Central and West Africa (Neuwinger 1996), although they are an effective poison in their own right. Although *N. glauca* is thought to be the true indigenous version of this species, this has been disputed (see Watt & Breyer-Brandwijk 1962). The Khoi herders at the Cape are recorded to have used a wide range of narcotic substances, particularly members of the Mesembryanthemaceae family, prior to the widespread adoption of imported tobacco (Gordon 1996; Van Wyk & Gercke 2000; Sobiecki 2002), but the southern African *Nicotiana* species are not amongst them.

*Nicotiana* species contain several toxic alkaloids, the most potent and well known of which is nicotine. Other toxic components include nornicotine, anabasine, cotinine, myosmine and anatabine (see Neuwinger 1996). Although never reported as an arrow poison in southern Africa, there is a case where an ostrich died from ingesting some
leaves of the *N. glauca* (Botha et al. 2011). Symptoms of severe nicotine poisoning in humans progress from a mucal burning sensation to disorientation, dry mouth, cold sweats, abdominal pain and finally to frothing at the mouth, cardiac arrest and suffocation (Neuwinger 1996).

**Ricinus communis** (Linne.) (castor bean plant)

Ricin derives from the castor bean plant *Ricinus communis* and is easily purified from castor-oil manufacturing waste (Musshoff & Madea 2009). The castor bean plant is widely used to control ticks on livestock and is taken for medicinal purposes, for example as a laxative or for muscle cramps (Bowles & Swaby 2006; Mawela 2008; Rana et al. 2012). The toxic alkaloids are present in the seed pulp rather than in the oil, and are water soluble (Wedin et al. 1986; Van Wyk et al. 2002; Mawela 2008). Ricin is known to be a very stable compound and does not degrade easily or quickly (Watt & Breyer-Brandwijk 1962). Ricin is a heterodimeric type-2 ribosome-inactivating protein (RIP), consisting of an A chain, a ribosome-inactivating enzyme (32 kDa) covalently connected by a disulfide bond to a galactose/N-acetylgalactosamine-binding lectin (34 kDa), also called the B chain (Musshoff & Madea 2009). The B chain is catalytically inactive, but serves to mediate entry of the A-B protein complex into the cytosol. The A chain is an N-glycoside hydrolase (Musshoff & Madea 2009). Purified ricin extracted from *R. communis* is highly toxic when injected, inhaled or ingested (Fredriksson et al. 2005). Ricin molecules consist of A and B chains, which are linked by a disulfide bridge. Both chains have a molecular weight of about 32 000. The B chain is responsible for binding to the cells of a victim, whereas the A chain causes the toxic action in the form of an RNA N-glycosidase (Fredriksson et al. 2005).

Symptoms of ricin poisoning include an initial burning sensation in the mouth and throat, followed by fever, headache, hypotension, abdominal pain, haemorrhaging and dehydration (Wedin et al. 1986; Musshoff & Madea 2009). Effects can be delayed for as long as 10–12 hours, but ultimately may progress to multisystem organ failure, with death occurring within 3–5 days (Wedin et al. 1986). Post-mortem findings include focal haemorrhage in the intestines, brain, myocardium and pleura. Lymph nodes, kidneys, and intestines may also demonstrate necrosis, haemorrhage and oedema (Musshoff & Madea 2009). Ricin can make a virile arrow poison as demonstrated by the ‘umbrella assassination’ of Georgi Markov, a Bulgarian dissident in 1978 (Sehep et al. 2009).

**Securidaca longepedunculata** (Fresen) (violet tree)

*S. longepedunculata* has been used as an additive in *Strophanthus* and *Adenium* arrow poisons in West Africa and Central Africa, and in southern Africa the shrub is used medicinally (Watt & Breyer-Brandwijk 1962; Neuwinger 1996; Van Wyk & Gericke 2000). The root is extremely poisonous and contains various saponines, salicylic acid and sterols (Neuwinger 1996) and is used to commit suicide in parts of Angola (Watt & Breyer-Brandwijk 1962). Extracts of the root cause cytotoxic tissue damage of internal organs (Maxwell et al. 2007). The root bark produces vomiting and purging.