POISONS AND POISONING AMONG THE ROMANS

by

Elizabeth Jennifer Richards
B.Sc., University of Victoria, 1979
B.A., University of Victoria, 1992

A Thesis Submitted in Partial Fulfillment of the
Requirements for the Degree of

MASTER OF ARTS

in the Department of Classics

ACCEPTED
FACULTY OF GRADUATE STUDIES
[redacted]
DEAN
DATE 17 May 94

We accept this thesis as conforming
to the required standard

[redacted]
Dr. John P. Oleson, Supervisor (Department of Classics)

[redacted]
Prof. K. Bradley, Departmental Member (Department of Classics)

[redacted]
Dr. Claude Spino, Outside Member (Department of Chemistry)

[redacted]
Dr. Nancy Turner, External Examiner (Environmental Studies)

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University of Victoria

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Supervisor: Dr. John P. Oleson

ABSTRACT

The purpose of this thesis is to explore poisons and poisoning in Roman antiquity — to find out what poisons were available and possibly used, to try to establish the identity, toxicity, and effectiveness of orally administered toxic substances, and to discuss the social and political context of intentional poisoning. The best sources of information on the poisons themselves are the medical and pseudo-scientific writers. The Roman historians and biographers are useful for their accounts of alleged incidents of intentional poisoning, but their reliability is very difficult to establish. Modern sources of toxicological information were used in an attempt to understand the toxicity of the poisonous substances. A variety of poisons was available to the Romans, but it is unclear how often and to what effect poisons were used.

Chapter I, the introduction, elaborates on the focus of the thesis — the types of poisons and poisoning included and excluded and the main chronological period. Methods and some of the problems in researching the topic are mentioned. This chapter also includes a brief survey of some of the ancient sources and a summary of general toxicology.

Chapter II covers the botanical sources of poison. Brief surveys of climate and plant toxicity are followed by the main poisonous plants of

antiquity — hemlock, aconite, the nightshade family, autumn crocus, hellebore, poppy, yew, and a particularly toxic mushroom.

Chapter III explores metallic (with a focus on lead) and animal sources of poison. It also covers substances that were thought to be poisonous in antiquity but were probably not.

Chapter IV examines the remedies of the ancient world and also ways of avoiding being poisoned. The ancients were aware of the most important and effective treatment for oral poisoning, but they also believed in the power of amulets and talismans as preventatives.

Chapter V deals with alleged motives for poisoning, ways of acquiring and administrating poison, and superstitions and theories surrounding poisons and poisoning. This chapter also contains a brief summary of the thesis.

Examiners:

[redacted]

Dr. J.P. Oleson, Supervisor (Department of Classics)

[redacted]

Prof. K.R. Bradley, Departmental Member (Department of Classics)

[redacted]

Dr. C. Spino, Outside Member (Department of Chemistry)

[redacted]

Dr. N. Turner, External Examiner (Environmental Studies)

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ACKNOWLEDGEMENTS

I would like to thank the University of Victoria for its generous financial support over the past two years. I am grateful to Dr. J. P. Oleson for his time, patience, and guidance, and to Prof. K. Bradley, Dr. C. Spino, and Dr. N. Turner for their penetrating criticism and helpful comments. I also wish to thank my colleagues for their moral support, good company, and unrelenting shower of relevant newspaper and magazine clippings. A special acknowledgement goes to Dr. Peter Smith, without whose gentle encouragement and incomparable teaching I may never have embarked on this project. Thanks also to Dr. Gordon Shrimpton for stepping into the breach and making my defence possible. As always, I owe a debt to my family for their continuing support and encouragement.

ABBREVIATIONS

Alex.	*Alexander* (Plutarch)
Alex.	*Alexipharmica* (Nicander)
An.	*Anabasis* (Xenophon)
Ann.	*Annals* (Tacitus)
Ant.	*Antonius* (Plutarch)
Carm.	*Carmina* (Horace)
Cato *Agr.*	Cato *De Agricultura*
Celsus *Med.*	Celsus *De Medicina*
Cic.	Cicero
adFam	*Epistulae ad Familiares*
Brut.	*Brutus*
Cael.	*Pro Caelio*
Clu.	*Pro Cluentio*
RhetHer	*Rhetorica ad Herennium*
Columella *Rust.*	Columella *De re rustica*
Comp.	*Compositiones* (Scribonius Largus)
Curt.	Quintus Curtius *History of Alexander*
Dig.	*Digest of Justinian*
Dio Cass.	Dio Cassius
Diod. Sic.	Diodorus Siculus
Ep.	*Epodes* (Horace)
Flor. *Epit.*	Florus *Epitome of Roman History*
Gell. *NA*	Aulus Gellius *Attic Nights*
Her.F.	*Hercules Furens* (Seneca the Younger)
Hist.Pl.	*Historia Plantarum* (Theophrastus)
HN	*Natural History* (Pliny the Elder)

Juv.	Juvenal
Larg. *Comp.*	Scribonius Largus *Compositiones*
Lucr.	Lucretius
Mart. *Ep.*	Martial *Epigrams*
MatMed	*Materia Medica* (Dioscorides)
Med.	*De Medicina* (Celsus)
Met.	*Metamorphoses* (Apuleius or Ovid)
Nic. *Alex.*	Nicander *Alexipharmaca*
Paul. Aeg.	Paulus of Aegina
Paus.	Pausanius
Pl. *Phd.*	Plato *Phaedo*
Pliny *Ep.*	Pliny (the Younger) *Epistulae*
Pliny *HN*	Pliny (the Elder) *Natural History*
Plut. *Ant.*	Plutarch *Antonius*
Pun.	*Punica* (Silius Italicus)
Rust.	*De re rustica* (Columella)
Sen. *Nat.*	Seneca (the Younger) *Natural Questions*
Stat. *Theb.*	Statius *Thebais*
Strat.	*Stratagems* (Frontinus)
Suet.	Suetonius
Calig.	*Gaius Caligula*
Claud.	*Divus Claudius*
Gal.	*Galba*
Ner.	*Nero*
Tib.	*Tiberius*
Tac. *Ann.*	Tacitus *Annals*
Theophr. *Hist.Pl.*	Theophrastus *Historia Plantarum*
Verg.	Vergil
Ecl.	*Eclogues*

G.	*Georgics*
Vitr.	Vitruvius
Xen. *An.*	Xenophon *Anabasis*

CHAPTER I: INTRODUCTION

Because modern technology can detect minute quantities of toxic substances in our bodies, and because access to poisons is restricted, we are essentially free from the fear of being deliberately poisoned. A far greater danger today is inadvertent poisoning by industrial and agricultural chemicals or accidental consumption of poisonous plants and household cleaning products. Concern about accidental poisoning is relatively recent; for most of human history the situation has been the reverse. In Roman times poisons were used as murder weapons, but compared to the situation today the Romans were relatively free from industrial poisoning. There is a possibility, however, that they were unintentionally exposed to a few toxic substances because of ignorance of the finer points of drug action and because of occupational hazards. Poisoning may have been popular in ancient Rome at certain times, perhaps because both the identity of the perpetrator and the cause of death could be concealed. The purpose of this study is to discover what poisons were available to the Romans and thus possibly used by them, and to try to determine the identity, toxicity, and possible effectiveness of the poisonous substances alluded to in ancient sources. I begin with the botanical poisons which are the most frequently mentioned source of poison in antiquity, probably due to the variety and accessibility of plants. The next chapter deals with animal, metallic, and imaginary or perceived poisons. A study of poisons would be incomplete without an examination of remedies, and a chapter on the treatments in antiquity has been included. The final chapter covers motives, acquisition and administration, and superstitions and theories surrounding poisons —

in short, people and poisons.

The primary focus of this paper is the Roman period from approximately 100 B.C. to A.D. 100, mainly because this period offers the greatest abundance of sources. Naturally, it is necessary to drift beyond these boundaries, since it is impossible to examine this period in complete isolation. There are excellent sources of information dating from the fourth century B.C. to the seventh century A.D. which both influenced and reflect the Roman practices of the period stated above.

The kinds of poisoning included in this study are intentional and unintentional poisoning by mouth, although topical poisons are covered briefly. Occupational poisoning is brought in where relevant to the main topic. Excluded are the bites and stings of venomous animals and poison-tipped arrows and spears. These limitations on the topic were imposed both because of my own personal interest and because of the necessity of reducing the topic to a manageable size.

The study of poisons and poisoning in Roman antiquity presents some difficulties. No modern book analyses the topic. Occasionally, however, books on related subjects, such as ancient medicine, contain a chapter on, or make reference to, drugs and poisons, or the topic is dealt with briefly in the introductions to books on, for example, general toxicology. Journal articles provide a slightly richer source of information, although the topic does not appear to be a popular one. Many ancient sources, while providing significant information, are prone to distortion and leave much unsaid. Ancient writers rarely wrote freely on the topic of poisons; such information was dangerous as general knowledge and deadly in the wrong hands. In addition, it was a topic that was susceptible to gossip and exaggeration. Nevertheless, by patching together information from a wide variety of both modern and ancient sources, I was gradually able to form a picture of Roman poisons and poisoning.

In spite of their reticence, the ancient sources do provide valuable, enlightening information. Pliny the Elder provides the greatest volume of material, but much of it is of questionable accuracy. Pliny combined encyclopedic zeal with little discrimination, and in his rush to write down everything he had ever seen, read, or heard, he often neglected to sleep; as a result he would sometimes doze off while working (Pliny *Ep.* 3.5.7-17). Raised in what is now Northern Italy, Pliny adopted the more severe, restrictive, and older codes of behaviour of his municipal governing class, in contrast to those adopted by the Roman court and aristocracy.[1] As a result, he is perceived as a narrow-minded and pedantic writer who "found time for many intellectual activities, but not often for second thoughts."[2] Although Pliny feels that the topic of poisons is a dangerous one, his work is full of references to poisons and their remedies. In general, Pliny's descriptions of plants do not arise from personal acquaintance with local flora, but are copied.[3] He recognized Theophrastus as a botanical expert and authority, but Pliny's attempts at plant description are less complete and less accurate than Theophrastus'.[4] Similarly, some of Pliny's descriptions of animals are so

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1 Reynolds (1986) 1.

2 Kenney (1982) 670.

3 Morton (1986) 91.

4 Morton (1986) 89 & 91.

vague and inaccurate that identification is difficult, if not impossible.[5] Fortunately, only five of the poisonous animals he describes are important to this discussion, and his information on poisonous plants can be fleshed out by Theophrastus and Dioscorides.

Dioscorides was a contemporary of Pliny. The two authors appear to have been unaware of one another, yet they used a common source for medicinal plants, Sextius Niger.[6] Dioscorides was a military physician who wrote a treatise on the medicinal uses of plants, animals, and minerals. He had studied the pharmacy of his time and is praised for his detailed observations, sound judgment, and freedom from superstition.[7] His work complements Pliny's and provides a valuable source of data.

Nicander's poem, *Alexipharmica*, devoted to poisons and their remedies, might, at first glance, be thought the best source for information on toxic substances in antiquity. Nicander does provide much useful material, but many of his 'facts' are questionable and some must be dismissed altogether. Nicander's dates are uncertain, but they range from the first half of the third century to the last third of the second century B.C.[8] His source was a prose treatise by Apollodorus (early third century B.C.).[9] Nicander himself seems to have been unacquainted with his subject, and he managed to mutilate Apollodorus'

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5 Bodson (1986) 101.

6 Scarborough (1986) 64 & 62.

7 *OCD* (1970) 353-4.

8 Easterling (1985) 602.

9 Easterling (1985) 602-3.

facts in transmission.[10] Nicander is criticised for using obscure Greek and mixing in absurd fable;[11] indeed he is guilty of "studied obscurantism," of using language that is "archaic and elaborate, full of lexical rarities," and of a lack of concern for "scientific exactitude."[12] Despite his numerous faults, Nicander is an important source of pharmacological and toxicological information for the Roman world.[13]

Other useful sources for information on poisons and poisoning are Celsus, Scribonius Largus, and Paulus of Aegina. Celsus was a layman who wrote about medical matters around the time of Tiberius (14-37 A.D.).[14] Largus, a physician, produced his work during the reign of Claudius and even accompanied the emperor to Britain.[15] Largus lists specific poisons, their symptoms, and their remedies. Paulus, another medical writer, is a considerably later source, dating between 600 and 650 A.D. He includes information about poisons and drugs in a larger work on medicine.

Apart from the medical and pseudo-scientific writers of antiquity, the works of historians, biographers, rhetoricians, and satirists contain useful material on poisons and poisoning. The two main sources for the period when poisoning — real or imagined — is most frequently mentioned, the first century A.D., are Suetonius and Tacitus. These two

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[10] Scarborough (1978) 355, fn. 15.

[11] Houghton (1885) 23.

[12] Easterling (1985) 603.

[13] Scarborough (1979) 83.

[14] *OCD* (1970) 218.

[15] Baldwin (1992) 74 & 76.

were contemporaries in the last quarter of the first century and the first quarter of the second century A.D. Unfortunately, Suetonius, a biographer of the Caesars, lacks a critical approach to his sources, tends to mix rumour with fact, and enjoys scandal.[16] On the other hand, he is also "one of the most informative and vivid sources for the history and society of the early empire."[17] Clearly, caution is necessary when using this source. The historian Tacitus collected his material from a wide range of sources, including friends who could provide eyewitness reports, writers who had lived in earlier times, and archives.[18] Although Tacitus ascribes motives and thoughts without sufficient evidence, he is not guilty of inventing or suppressing facts; of all the Roman historians, he is considered the most accurate.[19] Most of the ancient sources used in this paper are criticised for one reason or another by modern scholars. Nevertheless, our understanding of the past depends on the manuscripts and artifacts that survive. We must work with what we have and, while examining the data, keep their shortcomings in mind.

I would like to stress at this point that I do not treat any reference to poison or poisoning in the ancient sources as absolute proof of, for example, the identity of a poison or that a certain person was poisoned. Much of the information that we have has been filtered through many hands and cannot have escaped completely from alteration or embellishment. Furthermore, some of the ancient authors were working

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[16] Kenney (1982) 663 & 662.

[17] Wallace-Hadrill (1983) vii.

[18] Kenney (1982) 648-9; Mellor (1993) 32-3.

[19] Mellor (1993) 40.

from second-hand material rather than first-hand experience, while others had little knowledge of the particular field they were tackling. Still others enjoyed rhetoric and exaggeration for effect; therefore, the authenticity or accuracy of ancient references can be debated almost endlessly.

There are several Latin terms concerned with poisons and poisoning. The first step in my research was to develop a word-list. I began by consulting the *OLD* and, in the course of reading general background material, I discovered many more terms. The words used to refer to poisons in general are: *venenum* (poison); *venenatus* (poisonous); *venenare* (to poison); *venenifer* (poisonous).[20] The terms *veneficium* and *veneficus* refer to poison in a secondary sense; their primary meanings have to do with sorcery and the magical arts. *Virus* refers mainly to the poison of venomous animals, and *toxicum* means a poison, but especially the poison used on arrows. Other general terms are *malum medicamentum* (harmful drug or poison) and *herba nocens* (harmful or injurious plant). Specific plant poisons are: *cicuta* (hemlock); *aconitum* (monkshood); *strychnon, trychnon, solanum,* and *dorycnion* (all members of the nightshade family); *hyoscyamus* (henbane); *mandragoras* (mandrake); *ephemeron* (autumn crocus); *elleborus/veratrum* (hellebore and false hellebore); *opium* (opium); *taxus* (yew); and *boletus* or *fungus* (mushroom) plus an adjective to indicate a poisonous quality. Animal sources of poison are: *cantharis* (blister beetle); *buprestis* (a poisonous

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[20] In addition to meaning poison, *venenum* also refers to a potent herb, plant, or other substance used in medicine and magic; the verb *veneno* has overtones of witchcraft or enchantment. I have tried to identify the context of the instances of these words and use only those which seem to refer to poisons and poisoning.

beetle); *salamandra* (salamander); and *pityocampa* (a caterpillar). Miscellaneous or perceived poison sources are: *lepus marinus* (sea hare); *sanguis tauri* (bull's blood); *aqua Stygis* (Stygian water); and certain types of honey (*mel*).

I used the PHI CD-ROM and the Ibycus program to search some of these terms in Latin literature. *Venenum, venenatus, venenare* and *venenifer*, searched under the root *venen-*, produced the largest number of matches (approximately 1500). Not all instances of the words were useful — poison terminology was sometimes used metaphorically or in titles, but for the most part the list provided interesting or useful information. The searches for the specific poisons yielded less than 100 matches for each word. Any words that turned out to be the product of conjecture due to textual problems were not used as evidence for poison in this paper.

A brief survey of toxicology is necessary before launching into a discussion of Roman poisons.[21] It is not always easy to define a poison. A poison may be thought of as a foreign substance which has no metabolic or beneficial role in the body, and which produces an adverse reaction. But the presence and severity of the reaction depends on dosage and, in fact, the dose can determine the actual toxicity of a substance. An overdose of a substance normally used by the body can have toxic effects. A classic example of the importance of dose is table salt. Sodium and chloride are both essential to the continuation of human life, but

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21 Explanations of poison and its action may be found in Pascoe (1983) and Glaister (1973) 509-12. A more detailed treatment of the field of toxicology is given by Doull (1980). The following survey of toxicology is derived from these sources.

sodium chloride taken in excess can produce toxic effects.[22] Paracelsus, in the early sixteenth century, realized the significance of dose when he wrote:

> All substances are poisons; there is none which is not a poison. The right dose differentiates a poison and a remedy.[23]

Besides dose, there are other factors which affect the toxicity of a substance. Poisons are absorbed more rapidly and thoroughly from an empty stomach than from a full one. The general health of the victim is also important, since a weak, ill, or malnourished person may die from a dose from which a healthier, heartier person would recover. The very young and the very old are more susceptible to poisons, and habituation leads to some degree of tolerance to certain toxic compounds.

Poisons may gain access to the body in a variety of ways. In decreasing order of effectiveness they are: intravenous injection and inhalation of gas or vapour; subcutaneous or intra-muscular injection; ingestion; and application to the skin or mucous membranes. The most common method of intentional poisoning in antiquity was oral administration. Injection of poison through the skin could be achieved with poisoned arrows and spears, but the skin normally provides a protective barrier to toxic substances unless it is cut or abraded. A poison must pass through five layers of epidermis before reaching the dermis, where it can diffuse into the circulatory system. Exposure to foreign substances by inhalation was a hazard faced by mine and metal

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[22] Pascoe (1983) 1.

[23] Wexler (1982) 5.

workers, but it is not my intention to cover occupational hazards in antiquity, which is another topic altogether.

In order to elicit specific symptoms, poisons usually need to be absorbed into the bloodstream. Some poisons may have a local effect, such as the immediate irritation, burning, or corrosion of caustic substances on the skin and in the gastrointestinal tract, while others target a specific site, such as the heart, nervous system, or blood. Inhaled poisons are rapidly and effectively absorbed from the lungs because of the high degree of pulmonary vascularization. Swallowed poisons may be absorbed easily from the stomach, and gastric churning improves the chances of the poison coming into contact with and being taken up by the stomach walls. The contents of the stomach eventually pass into the small and then the large intestine, both of which are excellent sites for the absorption of foreign substances. Once a poison has gained access to the bloodstream, it is carried to all the tissues and organs of the body.

The body is not without resources with which to combat an invasion of foreign substances. Mechanisms are in place to metabolize poisons — to decrease their toxic activity and to increase their excretion.[24] For the most part, this metabolism takes place in the liver and yields compounds that may be excreted via the kidneys, the bile, and the gastrointestinal tract. Although these are the main routes for the excretion of poisons, some toxic substances can also be eliminated through hair, nails, tears, sweat, expired air, and milk.

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24 There are a few chemical compounds which actually demonstrate an increased physiological effect and increased toxicity after undergoing metabolic change; see Pascoe (1983) 21.

CHAPTER II: PLANT POISONS

The botanical world has long been a rich source of drugs and poisons. Early humankind can only have learned the characteristics of plants by trial and error and by watching what wild animals ate or avoided (although the effect on animals may differ from the effect on human beings). Even after the advent of literacy, detailed information about poisons and dangerous drugs was probably handed down orally within a select group of people. Only a relatively small percentage of the population was literate anyway,[1] and such arcane and powerful information was likely to be guarded and restricted. The division between drug and poison is hazy; what has a therapeutic effect at a certain dose or on a particular species may have a poisonous, even deadly, action at a different dose or on another species. Vegetable poisons are easier to collect than animal poisons; snakes, scorpions, and other venomous animals must be found and pursued, they are tricky and dangerous to catch, and the extraction of venom takes skill and knowledge. Plants are sessile, and the leaves, roots, seeds, and flowers can be dried and preserved, or the oils can be extracted relatively easily.

Since climate and seasonal factors can influence the concentration of toxins in plants, the climate of the classical world is an important factor. The rich and varied assortment of plant life in the Mediterranean grows in a distinct climate.[2] The summers are sunny, hot, and dry; rain falls from October to April, and the winters are mild. Due to the drought-

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1 Harris hypothesizes that the level of illiteracy in the Roman Empire was above 90 percent. (1989) 22.

2 Comprehensive descriptions of the Mediterranean climate may be found in Polunin (1987) 1-4, and Polunin (1985) 12-3 from which sources this brief summary is taken.

like conditions in summer, plant growth occurs in the autumn, with the onset of rain, and in the spring. Annuals tend to flower in the autumn and spring, and most perennials, flowering in early spring, die off in the summer. Fungi appear with the rain in spring or autumn depending on the species.

What makes plants poisonous? The actual role of toxic substances in plants remains, for the most part, unclear. It is possible that these substances act as a form chemical defence; a toxic plant may be unpalatable, or worse, to herbivorous animals, and this characteristic may prevent it from being eaten. Toxic properties may also discourage other plants from growing nearby and crowding the poisonous plant, thus reducing its chances for survival. On the other hand, plant poisons may simply be by-products of metabolism.[3] The poisonous substances in plants can take the form of alkaloids, glycosides, proteins, resins, tannins, aldehydes, oxalates, or alcohols. The two largest groups of plant poisons are the alkaloids and glycosides, and of these two, the alkaloids were the most important botanical poisons in antiquity.

Alkaloids are basic (as opposed to acidic) nitrogen-bearing organic compounds that occur in 40 percent of plant families. They are capable of considerable physiological and pharmacological activity and can be found in the seeds, fruit, leaves, stem, root, or in any combination of these. The alkaloid-producing families relevant to antiquity are the buttercup family (Ranunculaceae), the nightshade family (Solanaceae), the lily family (Liliaceae), and the poppy family (Papaveraceae). Alkaloids

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[3] General discussions of the poisonous substances in plants may be found in Turner and Szczawinski (1991) 1-3 and 14-23; in Lewis (1977) 12-20; and in Claus (1965) 265-7 (alkaloids) and 92-4 (glycosides).

are uncommon in needle-bearing evergreens, with the exception of the yew (*Taxus*) in the family Taxaceae, and in the carrot or celery family (Apiaceae or Umbelliferae), with the exception of poison hemlock (*Conium*). Specific alkaloids are generally found within specific families (e.g., atropine in the Solanaceae), and a single species can contain various alkaloids with similar chemical structures. Many alkaloids affect the human nervous system, and some are structurally similar to human neurotransmitters; it is possible that they either block or mimic these substances and interfere with the normal functioning of the nervous system.

Of the other poisonous principles, glycosides occur in many plants, but not all are toxic. They are composed of one or more sugar molecules joined to one or more other components called aglycones; the aglycone is considered the toxic element. Cardioactive glycosides act directly on the heart muscle, and cyanogenic glycosides (yielding cyanide as one product of breakdown) inhibit the uptake of oxygen in the cells, causing asphyxiation. Polypeptides, chains of peptides which may be thought of as simple proteins, are responsible for the serious and often fatal poisoning associated with mushrooms; resins affecting the heart and circulation are found in the heather family (Ericaceae).

Plants usually share similar characteristics of toxicity at the generic level, although there are exceptions and often important differences at the species level.[4] In extreme cases, different genera within a shared family may have poisonous characteristics in common,

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[4] The following discussion of plant toxicity is derived from Doull (1980) 578-90 and Turner and Szczawinski (1991) 7.

or, of two closely related species, one may be poisonous and the other harmless. Sometimes the same poisonous principle shows up in plants that differ widely taxonomically and geographically. Environmental conditions, season, and age are all factors that can influence the concentration of poison in a plant. Not all poisonous plants are toxic to all animals — ruminants can often tolerate toxic plants better than animals with single stomachs. Deer eat yew and rhododendron, and rabbits are able to eat quantities of belladonna that are fatal to other animals. Quail eat poison hemlock seeds with impunity, but their flesh becomes poisonous; goats can eat colchicum, but the poison is transmitted to their milk.[5]

The toxic effects of plant poisons cover a broad range from irritation of the mucous membranes to disastrous disruption of cellular functions and biochemical processes.[6] The most common sign of poisoning by plants is gastrointestinal upset; nervous symptoms, such as convulsions, paralysis, or hallucinations, are next in frequency. Chewing and, by analogy, the grinding, pounding, or rubbing often employed in the preparation of a drug or poison can affect the toxicity of a plant; some highly toxic seeds pass harmlessly through the gut if they are swallowed whole (the intact seed coat prevents the escape of the poisonous principles). Upon ingestion, a plant toxin may irritate the inside of the mouth, the stomach, and the intestines; absorption of toxins can take place anywhere within the gastrointestinal tract. After a poison is absorbed from the gut into the circulatory system, it passes

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5 Stevens (1990) 45 & 58.

6 The following description of the toxic activity of plants is from Doull (1980) 578-90.

through the liver, where, depending on the toxin, it can destroy hepatic tissue. Within the circulatory system, where some toxins cause red blood cells to rupture or interfere with their ability to carry oxygen, the poison has access to all the body's organs. Some poisons cause heart failure, others dilation of the pupil, and others fetal deformation. Nervous symptoms take either an excitatory or depressive form, or can be manifested in paraesthesias or hallucinations.

Hemlock is probably the best known of the ancient plant poisons. As the poison of choice for executions in ancient Greece, its most famous victim was Socrates (Pl. *Phd.* 117-8). After Seneca opened his veins but failed to die, he drank hemlock, which must have facilitated his end (Tac. *Ann.* 15.64). Poison hemlock (*Conium maculatum*) is a member of Apiaceae or Umbelliferae, the carrot or celery family, and should not be confused with the poisonous water hemlock (*Cicuta* spp.), the coniferous hemlock tree of the genus *Tsuga*, or the poisonous ground hemlock, a shrub of the genus *Taxus*. Poison hemlock (*Conium maculatum*) is thought to be the same plant referred to in antiquity as *cicuta*; there are only two species in the *Conium* genus — one is found in southern Africa and the other is native to Europe and Asia.[7] Poison hemlock now grows worldwide; in Canada it is considered a weed of waste ground and roadsides.[8]

Poison hemlock is a tall (up to 2-3 metres), many-branched, leafy plant; its stem is smooth, hollow, and marked with purple spots; the

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7 Clark (1973) 328. André (1985) 66 agrees that the *cicuta* of antiquity is *Conium maculatum.*

8 Clark (1973) 328; Turner and Szczawinski (1991) 129; Hardin (1974) 108-9.

leaves resemble carrot or parsley leaves, and its small white flowers, which occur in umbrella-like clusters, have a musky or mousy odour; the root is carrot-shaped, and the fruit is round, small, and ribbed.[9] The poisonous principle is the alkaloid coniine, which is concentrated in the fruit and root, but the whole plant is poisonous. The concentration of coniine varies with age and climate; the young leaves are more toxic before the plant has flowered, and very sunny weather increases the alkaloid content in the whole plant. In addition, the alkaloid concentration in the fruit decreases with age; the green fruit contains about three times as much coniine as the ripe, yellow fruit. Poisoning in modern times has occurred by mistaking hemlock leaves for parsley, the seeds for anise, and the roots for carrots.

After a brief period of stimulation, coniine has a depressive action on the nervous system, resulting in gradual paralysis, which, when it reaches the respiratory muscles, causes death.[10] The muscles weaken painfully, but the mind remains lucid. Symptoms appear 1-3 hours after ingestion and may include vomiting, diarrhea, loss of coordination, dilation of the pupils, a feeling of cold in the limbs, difficulty in speaking, coma, and death after several hours. The fatal dose of coniine is believed to be 130 mg (or approximately 2 to 5 g of the actual plant) for the average human being (usually considered an 80 kg male); children have died from eating the root. There is no antidote — the only hope is to try

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[9] Descriptions of the natural history and toxicity of poison hemlock may be found in Clark (1973) 328; Hardin (1974)108; and Turner and Szczawinski (1991)128-9, from which sources this description is derived. Claus (1965) 16, deals with the variations in alkaloid content in the fruit.

[10] Details of the effects and symptoms of hemlock poisoning are from Kingsbury (1965) 65; Jensen (1970) 122; Stevens (1990) 44; Turner and Szczawinski (1991)129-30.

to reduce the amount of time that the poison remains in the body by means of emetics and cathartics, and to treat the symptoms.

Despite its fame, Plato's account of the death of Socrates is an inaccurate and sanitized version of the symptoms and effects of hemlock poisoning (*Phd.* 117-8). Plato did not actually witness Socrates' end; his description is based on secondhand information. He may have wished to describe Socrates' death as peaceful, dignified, and fitting for a great man. The poison was brought to Socrates in a cup. After drinking it, he was to walk around until his legs felt heavy and then lie down. Socrates drained the cup cheerfully and quietly, walked about until his legs felt heavy, then lay down. The man who gave him the poison began pinching Socrates' leg, starting at his foot, to check for sensation — there was none. He demonstrated how Socrates was becoming cold and rigid, and he said that when the poison reached his heart he would die. Socrates appears to have had his face covered while he was lying down. He uncovered his face, said a few words, re-covered his face, and, after an unspecified period of time, he quietly died.

According to Plato, Socrates drank the bitter and foul-tasting draught without a grimace or any sort of reaction. Plato's description of the ascending lack of sensation is puzzling, as anaesthesia is not a feature of coniine poisoning.[11] He seems to describe the gradual paralysis and feeling of cold but avoids any mention of retching, vomiting,

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[11] Ober (1979) 268. Other drugs or poisons might have been mixed with the hemlock. G. Glotz, in the *Dictionnaire des Antiquités grecques et romaines*, Daremberg & Saglio, t.3, 1re p., p. 863 cited by Grisé (1982) 110, n. 130, writes that the Athenian formula for hemlock was actually a mixture of hemlock and all kinds of toxic, narcotic, and sedating substances, whose effect was certain, fast, and painless; the dose was 1 drachma (4.36 g). Aconite can cause numbness of the skin and extremities (Socrates' lack of sensation?), and opium has a sedating effect (Socrates' serenity?), but it is difficult to predict the pharmacological action of several different drugs mixed together.

diarrhea, or distress of any kind. Socrates' mind remains clear to the end — a characteristic of hemlock poisoning. Throughout his ordeal, Socrates appears calm and unconcerned. It is possible that Plato chose to ignore the nastier symptoms of hemlock poisoning in order to allow Socrates to die with dignity; on the other hand, perhaps the draught did contain a sedative or narcotic.

Descriptions of the hemlock plant in antiquity are generally incomplete. Theophrastus describes its hollow stem and leaves resembling those of parsley (*Hist.Pl.* 6.2.9; 7.6.4). Pliny mentions the plant's smell and height (*HN* 25.151), and Dioscorides comments on the smell, white flowers, and seeds, which resemble anise (*Mat.Med.* 4.79). Theophrastus believed that the best hemlock grew in cold and shady places (*Hist.Pl.* 9.15.8; 9.16.8), although the coniine concentration actually increases with exposure to sunlight,[12] while Dioscorides felt that the strongest came from Crete, Megara, and Attica (*Mat.Med.* 4.79). Theophrastus says that the poison is concentrated in the roots (*Hist.Pl.* 9.8.3), while Pliny states that the seeds and leaves are dangerous (*HN* 25.151-2). Nicander writes that the victim suffers from ataxia, has trouble breathing, and his extremities grow cold (*Alex.* 186-94); Pliny and Dioscorides both attribute death to the chilling properties of hemlock (*HN* 25.152; *Mat.Med.* 4.79). Pliny says that a second characteristic of hemlock is that it causes death by thickening the blood — a process which causes spots to appear on the body (*HN* 25.152-3).

The remedies for hemlock poisoning in antiquity concentrated on counteracting the perceived cooling properties of the plant. Thus

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[12] Turner and Szczawinski (1991) 129.

warming substances were given, such as wine (*HN* 25.152; *Mat.Med.* 4.79), pepper (Theophr. *Hist.Pl.* 9.20.1; Nic. *Alex.* 195-201), or frankincense (Theophr. *Hist.Pl.* 9.20.1). Grape syrup and warm milk were also suggested by Nicander (*Alex.* 205-6). Hemlock was also used medicinally as a medicament to remove scabs on sheep and cattle (Columella *Rust.* 7.5.8), to treat local inflammation of the skin (Pliny *HN* 26.121), and as one ingredient of a concoction for protruding navels (Celsus *Med.* 6.17). Since the bad smell and taste of hemlock must have been difficult to disguise in antiquity, it is unlikely that *Conium maculatum* was the toxin of choice in intentional poisoning. It was better suited for execution, or suicide under compulsion.

Aconite was one of the strongest plant poisons available to the classical world. Commonly called monkshood or wolf bane, aconite belongs to the Ranunculaceae, or buttercup family — a primitive family whose members are often bitter and poisonous.[13] The actual species of aconite that existed in antiquity is unclear, but although the different species in the genus *Aconitum* may vary in their chemical make-up, they are all considered poisonous.[14] The tuberous root of aconite is considered one of the most toxic plant products known, and monkshood is thought to be the most poisonous plant growing in Great Britain. Aconite species grow throughout Europe and in a variety of habitats. The plant is a leafy perennial that can grow to more than a metre high. The leaves consist of narrow, pointed lobes, and the flowers, ranging from

[13] Polunin (1987) 65-6.

[14] The following physical description and natural history of aconite is derived from Turner and Szczawinski (1991) 204; Fatovich (1992) 309-10; and Hardin (1974) 52. Claus (1965) 331 discusses the time at which the alkaloid concentration is at its peak.

white to yellow to blue, are topped with a hood-like structure. All parts of the plant are poisonous, but the areas of concentration of the toxin (in decreasing order) are the root, the flower, the leaf, and the stem. The main poisonous principle is the alkaloid aconitine, which is at its highest concentration during flowering, although afterwards the concentration remains high. Modern poisonings occur by mistaking the root for horseradish, the leaves for parsley, or by drinking an infusion of aconite as a herbal cure.

The symptoms of aconite poisoning usually appear within the first hour after ingestion and begin with tingling and a feeling of numbness in the lips, mouth, and throat; numbness may spread to the extremities and over the entire surface of the skin.[15] The heart initially slows down and then becomes more excitable and uncoordinated. The disturbance of the gastrointestinal tract results in salivation, nausea, vomiting, and diarrhea. Respiration becomes slower, and the skin becomes cold and clammy. The victim feels cold and weak, lacks coordination, has a headache, and experiences vertigo. Vision may be disturbed. Because of the gradual paralysis of the cardiac muscle, the heart may stop without warning. The victim can die within six hours, or death may take as long as four days; a large dose, however, can kill instantly.

Aconite was once used medicinally but has now fallen out of favour; the beneficial and deleterious doses are too similar and the concentration of aconitine in each plant is variable and cannot be predicted accurately. Monkshood was used to slow and reduce the force

[15] The pharmacological effects and symptoms of aconite poisoning are derived from Martindale (1982) 1674; Mack (1985) 519; Fatovich (1992) 310; and Turner and Szczawinski (1991) 204-5.

of heartbeats, and as a diaphoretic (a sweat-producing agent used to reduce fever). Rubbed on the skin in the form of a liniment it causes tingling and numbness and was used to treat neuralgia and rheumatism, but enough aconite can be absorbed through the skin to cause serious poisoning. Aconite preparations are sometimes prescribed by homeopathic practitioners with serious and sometimes fatal results. The fatal dose of aconite is only 1 gram (for the average person) of the actual plant, and simply picking the flowers can numb the fingers. Since there is no antidote for aconite poisoning, modern physicians can only treat the symptoms. Emptying the stomach using an emetic (usually Ipecac syrup, derived from a plant containing the alkaloid emetine) or by gastric lavage, followed by a dose of activated charcoal and a cathartic helps rid the body of the offending substance, reduces its chances of absorption, and hastens its passage through the intestinal tract. This treatment is followed by monitoring the heartbeat and administering heart stimulating drugs if necessary. The patient is kept warm and given fluids and electrolytes.

The symptoms and effects of aconite poisoning were observed in antiquity. Pliny noted the speedy effect of this poison[16] as well as its cooling properties, which he thought were the cause of death (*HN* 29.75). Nicander described the effect of aconite on the mouth, the ensuing headache, the effect on the stomach, and disturbed vision (*Alex.* 16-29). Theophrastus was correct in thinking that the poison in aconite was found in the root but dangerously incorrect in saying that the leaves and fruit were harmless (*Hist.Pl.* 9.16.4). He noted that aconite grew

[16] *constat omnium venenorum ocissimum esse aconitum* ... (*HN* 27.4).

everywhere but especially liked rocky ground, and that animals avoided eating it (9.16.4). He appears to have been misled in thinking that in order to be effective aconite must be processed in a particular manner and that it could be manipulated so as to have the quality of latent fatality — death could be arranged to take place from two months to two years after ingestion (9.16.5). He was correct, however, that aconite has no antidote (9.16.5).

The Roman physician Scribonius Largus describes some of the symptoms of aconite poisoning. He writes that a drink of aconite will make the body heavy and uncomfortable, hurt the gullet, and affect the heart (*Comp.* 188). These symptoms are followed by much cold sweat, intestinal troubles, headache, and dizziness. He claims that it is characteristic of this poison to make the limbs livid. Dioscorides' account of aconite is confusing, as he refers to two different plants, both called aconite; he describes no symptoms of human poisoning, saying only that the plant is used for poisoning bait set for wolves, panthers, and other animals (*Mat.Med.* 4.77-8). Pliny also mentions that leopards are caught with meat rubbed with aconite (*HN* 8.100).

An explanation of the existence of aconite is given by both Pliny and Ovid. They relate a myth that foam flew from the mouth of Cerberus as he was dragged from the underworld by Hercules and turned into aconite where it fell on the ground; thus it grows in Heraclea in Pontus, the location of the entrance to the underworld (*HN* 27.4-5; *Met.* 7.408-16). Pliny gives two explanations for the name: since it grows in rocky and relatively soiless conditions, it derived its name from the Greek *akonitos* meaning "without dust"; alternatively, its ability to deliver speedy death was similar to the quick death delivered by iron sharpened with a whetstone, or *akone* in Greek (*HN* 27.10). Ovid writes that the plant is called aconite because it flourishes on hard rocks (*Met.* 7. 418-9). Theophrastus thought it was named for the village of Akonai where, among many other places, it grew (*Hist. Pl.* 9.16.4). Nicander says that aconite flourishes in the Aconaean mountains (*Alex.* 41-2).

Although at the present time there is no known antidote for aconite poisoning, the ancients believed in numerous and various remedies. A few of Nicander's remedies are gypsum in wine with wormwood added, spurge-olive and rue in warmed vinegar and honey, or the meaty and undiluted broth of a fowl (*Alex.* 43-73). Pliny suggests garlic (*HN* 20.50), pounded rue leaves taken in wine (20.132), pure wine (23.43), oil of balsam in milk (23.92), mulberry juice in wine (23.135), tripe broth and veal suet (28.161), or warmed sheep's milk (29.105). A few of Scribonius Largus' recommendations are rue drunk with wine, chicken or beef broth, and warmed vinegar and honey (*Comp.* 188).

The use of aconite as a poison appears to have been common knowledge at Rome; references to it appear in a wide range of literature. Although Juvenal wrote satirically and exaggerated for effect, his references to aconite indicate that this particular poison was well known. A man allegedly disposed of several uncles with aconite (Juv. 1.158), and a woman killed her children (6.638-9). Juvenal adds that the poor man need not fear poisoning by aconite; earthenware never holds such a potion but jewelled and golden cups do.[17] Ovid speaks of aconite as a substance used to eliminate family members in times past (*Met.* 1.146-7),

[17] *sed nulla aconita bibuntur fictilibus* (Juv. 10.25-6).

and says that Medea used aconite in her attempt on the life of Theseus (*Met.* 7.406-7).

The family Solanaceae, the nightshade family, harbours several poisonous members belonging to different genera. Common food plants, such as potato, tomato, pepper, and eggplant, also belong to this group.[18] The poisonous plants which may have been known in antiquity are deadly nightshade (*Atropa belladonna*), henbane (*Hyoscyamus*), thorn-apple (*Datura stramonium*), and mandrake (*Mandragoras*). Atropine and related alkaloids are the poisonous principles in all these plants, and they have similar effects on the human body. Confusion reigns in regard to specific identification of these plants in antiquity, but since all four of the above genera grow in the Mediterranean and were referred to in one form or another in antiquity as poisons, they do merit consideration and an attempt at sorting them out.

Deadly nightshade, or belladonna, is a tall (up to 1.5 metres), branching, herbaceous (not woody) perennial with large oval leaves placed alternately along the stems.[19] The leaves sometimes occur in pairs of one large and one very small leaf. The flowers are solitary, bell-shaped, and brownish purple; the berries are shiny, black or purple, and they rest on a spreading bed of hardened sepals. Belladonna is a native of central and southern Europe and Asia Minor; in the Mediterranean it grows, for the most part, in the mountains in either woods or clearings.

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18 The leaves of these plants are toxic to varying degrees, as are the sprouts and green tubers of potatoes. Turner and Szczawinski (1991) 263.

19 Hardin (1974) 136; Huxley (1977) 127-8; and Turner and Szczawinski (1991) 183-4 are the sources for this description of deadly nightshade.

The whole plant, especially the berries, contains the alkaloids atropine, hyoscyamine, and hyoscine (or scopolamine).[20]

Belladonna initially excites but then depresses the central nervous system, inhibits the secretion of saliva and sweat, and slows down the movements of the stomach and intestines.[21] The symptoms of belladonna poisoning include abdominal pain, hot and dry skin, rapid pulse, dilation of the pupils, and dry mouth. Vomiting, dizziness, ataxia, rapid breathing, and hallucinations may occur; in severe cases circulatory and respiratory failure are followed by death. Fatal poisonings are not common. Trying to identify deadly nightshade in the ancient authors is a disheartening task. According to the *Oxford Latin Dictionary*, most of the vocabulary relating to the nightshade family is vague and interchangeable. Because of the confusion of names for the members of this family, a discussion of the problems in the ancient sources will appear at the end of the Solanaceae section.

Henbane is native to Europe and grows on waste land, in rocky places, and often close to buildings.[22] The plant is coarse and hairy with a faintly unpleasant smell. The leaves are simple and generally ovoid with blunt, irregular teeth along the margins. The tubular or bell-like flowers range from greenish yellow to pale purple and are often purple or green

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[20] Martindale (1982) 289-91, contains a good description of atropine and its effects; hyoscyamine and hyoscine are considered to have similar effects.

[21] Atropine, hyoscyamine, and hyoscine are called anticholinergic agents as they interfere with the neurotransmitter acetylcholine, which transmits impulses between nerve fibres; anticholinergic agents affect the parasympathetic nervous system which controls involuntary muscle activity such as the beating of the heart, gastrointestinal movements, and the activity of the glands. (*Oxford Comp. Med* (1986) 3,117,1008).

[22] Descriptions and discussions of henbane may be found in Hardin (1974) 140; Huxley (1977) 128; Polunin (1987) 164-5; and Turner and Szczawinski (1991) 197-8. Martindale (1982) 304-5 discusses the pharmacological properties of henbane.

in their depths. The whole plant is toxic, with the highest concentration of poison in the roots and seeds. The alkaloids hyoscine, atropine, and hyoscyamine are responsible for the toxic properties of this plant, but hyoscyamine is the principal toxic constituent in mature plants.[23] Hyoscyamine and hyoscine are related to atropine (all three are considered tropane alkaloids[24]); in fact, the process of heating converts hyoscyamine to atropine. Henbane causes dizziness, hallucinations, confusion, and excitement, along with symptoms similar to those of deadly nightshade. The dried and powdered hyoscyamus leaf has an unpleasant odour and a slightly bitter taste. The treatment for henbane poisoning is the same as that for all plant poisons dealt with so far; empty the stomach, administer activated charcoal and a cathartic, and monitor the symptoms.

Celsus considered henbane both a remedy and a poison; he recommended that it be taken internally as a pain-killer and soporific (*Med.* 5.25.2; 3.18.12), but he also suggested that it could be used externally (usually in combination with other components) for general joint and sinew pain (3.27.2c; 5.18.29). For ear problems the juice of henbane could be used by itself (*Med.* 6.7.2d). Celsus also lists remedies for hyoscyamus poisoning — hot wine and honey or milk, especially ass's milk (*Med.* 5.27.12b). Scribonius Largus writes that a henbane draught produces heaviness in the head and stretched veins (*Comp.* 181). Those who have drunk it wander in their minds and argue; afterwards they fall asleep and have no sensation (*Comp.* 181). After being forced to vomit,

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23 The immature plants contain more hyoscine (Duke (1985) 241).

24 Cocaine also belongs to the tropane alkaloids.

the victims should be given ass's milk or any other kind of milk (*Comp.* 181).

Pliny recorded both the effects of henbane on the head and its various medicinal uses; he also described four different kinds of henbane which cannot all be securely identified. The first variety he describes as having almost-purple flowers, black seeds and a spiny calyx (*HN* 25.35).[25] The next two varieties are not described sufficiently to make an attempt at identification, but Pliny may have been referring to different species of henbane such as *Hyoscyamus albus* and *H. aureus* which grow throughout the Mediterranean.[26] Of the effects of the first three kinds of henbane, Pliny says that they cause dizziness and insanity (hallucinations, confusion, and excitement may have been perceived as madness in antiquity), while the fourth is used for medicinal purposes (*HN* 25.35-6). Pliny felt that henbane had the nature of wine in that it affected the head and the mind; he thought that, on the whole, henbane was a risky, ill-considered drug, and even the oil poured into the ear would affect the mind (*HN* 25.36-7). In spite of his feelings towards henbane, Pliny recorded many medicinal uses in addition to remedies for poisoning. He says that henbane root chewed with vinegar is a good remedy for teeth problems (*HN* 25.165), the juice is good for coughs (26.27) and the juice or seed is useful for gout (26.100). The suggested remedies for poisoning are garlic (*HN* 20.50), nettle and broth of boiled turtle (22.31), and milk, either a woman's (28.74) or an ass's (28.158).

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[25] Turner and Szczawinski (1991) 197 describe the flowers of henbane as sometimes mauve "marked with a network of dark purplish veins. In fruiting, the calyx grows around the fruit and hardens into five pointed spines."

[26] Huxley (1977) 128 and Polunin (1987) 164-5.

Dioscorides describes three varieties of henbane. The first has almost-purple flowers, black seeds, and a spiny calyx; the second has yellowish flowers and pale yellow seeds, but both cause madness and act as soporifics (*Mat. Med.* 4.69). The third variety is suitable for medicinal purposes; it has white flowers and seeds and grows near the sea and around buildings (4.69). Many of the medicinal uses are the same as Pliny's (such as pain-killer and ear and toothache remedy), but he warns of its effect on the mind if eaten in quantity. Dioscorides records no remedies for henbane poisoning. Nicander, on the other hand, does not describe the henbane plant, nor does he discuss medicinal uses but treats it only as a poison. He warns against ingesting it and, interestingly, mentions that children who have begun to walk might chew on the plant to ease teething pains (*Alex.* 415-22).[27] Nicander seems to assume that henbane was a well-known plant since he identifies it by name only. His remedies include milk, nettle seeds or leaves, garlic, and a variety of herbs and vegetables (*Alex.* 423-32).

The third of the four solanaceous plants is thorn-apple or datura (*Datura stramonium* and related species). Due to its similarities with henbane, this plant could easily have been, and probably was, confused with it in antiquity. At present, datura grows on both waste and cultivated land throughout the Mediterranean.[28] The plant is tall and branching with a disagreeable smell and large leaves toothed along the

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[27] This seems to confirm that the plant grew near habitations as Dioscorides mentioned.

[28] This description of datura is from Duke (1985) 161-2; Huxley (1977) 128; Polunin (1987) 166; and Turner and Szczawinski (1991) 118-9. Martindale (1982) 311 discusses the pharmacological properties of datura.

margins. The white or occasionally purplish flowers are trumpet or funnel-shaped, and the distinctive fruit, green and spiny (similar to horse chestnuts), contains numerous black seeds. The whole plant is poisonous, particularly the seeds and leaves, and contains hyoscyamine as its principle alkaloid but also hyoscine and atropine. In this respect also it is similar to henbane and produces similar symptoms of poisoning, such as dry mouth, dilated pupils, hot and dry skin, blurred vision, headache, nausea, hallucinations, dizziness, delirium, tachycardia, and sometimes death. The seeds have been used for both suicide and murder.[29] Treatment is the emptying of the stomach followed by activated charcoal and cathartic.

Mandrake, the last of the solanaceous plants, must be included (although it is not considered a serious poison) since the Digest includes it in its list of substances that dealers in cosmetics were not permitted to sell to people recklessly (*Dig.* 48.8.3.3). Mandrake grows in a low, flat, spreading rosette and has long, ovoid, wrinkled leaves.[30] The light blue to violet flowers are borne at the centre of the plant, as are the yellow or orange edible fruits that resemble ornamental plums. The thick and fleshy root, when it is forked, is said to resemble a human being. The plant grows in fields and on stony ground. Mandrake contains several alkaloids, among them atropine, hyoscyamine, and hyoscine with hyoscyamine as the principle alkaloid.

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29 Duke (1985) 161.

30 A description of *Mandragora officinarum* may be found in Huxley (1977) 165-6 and Polunin (1987) 128. It is not included in Turner and Szczawinski (1991) as a dangerously poisonous plant, but, in addition to the medicinal properties listed in Duke (1985) 292, it is also considered a poison.

There is a long history of superstition surrounding mandrake. Theophrastus wrote that three circles should be drawn around the plant with a sword, and the harvester should face west; for a second piece he should dance in a circle and say as much as possible about love (*Hist. Pl.* 9.8.8). Mandrake, when uprooted, was believed to give a shriek fatal to its gatherer; the solution was to tie a hungry dog to the plant and induce it to pull with the temptation of food.[31] Mandrake was used in the Middle Ages as an anaesthetic but was also associated with witchcraft. It was said to grow in places defiled by the execution of criminals. In later years the root was worn as a charm to encourage either love or fertility.

According to Theophrastus, the leaves of mandrake were used for wounds and the root for erysipelas (inflammation of the skin), gout, insomnia, and love potions (*Hist.Pl.* 9.9.1). Celsus refers to mandrake as one ingredient in soporifics and pain-killers (*Med.* 5.25.2; 5.25.3b), and Frontinus thought it fell somewhere between a poison and soporific (*Str.* 2.5.12). Dioscorides' description of mandrake is similar to modern descriptions; the leaves are long and narrow, the plant grows low to the ground, bears fruit, and has a large root (*Mat.Med.* 4.76). Unfortunately, he further distinguishes between two kinds of mandrake: white and black. The root was used for love potions, soporifics, pain-killers, and as an anaesthetic for cutting and cauterizing (*Mat.Med.* 4.76). The juice was useful in eye medicines, as an emmenagogue and abortifacient, and for ulcers, but fatal if taken in excess (*Mat.Med.* 4.76). Pliny mentions various external uses for mandrake such as in a salve for abscesses and ulcers (*HN* 26.145), with barley to draw out things imbedded in the flesh

[31] Thompson (1931) 58-63 covers the history of mandrake more fully.

(26.149), the juice as part of eye medicines (25.147), or for erysipelas (inflammation of the skin) (26.121). He also suggests that the seed taken in drink acts as a uterine purgative, and a pessary of the juice could bring on menstruation or expel a dead fetus (26.156). Pliny's description of the actual mandrake plant is not very helpful for the purposes of identification. He, like Dioscorides, claims that there are two kinds: white and black (*HN* 25.147-50). His version of the rites surrounding its collection sound much like a repetition of Theophrastus except he recommends keeping one's back to the wind. Mandrake was used as a soporific, but Pliny warns that drinking too much can be fatal (*HN* 25.150). Like Dioscorides, Pliny says that mandrake has an anaesthetic effect and is drunk before cuttings and stabbings, presumably those associated with surgery, to eliminate sensation.[32]

Based on the vocabulary used in the ancient literature, it is almost impossible to securely identify and distinguish among the members of the nightshade family; one can only make, at best, shaky guesses based on a few recorded symptoms and plant characteristics. The *Oxford Latin Dictionary* is able to shed little light on the subject. The words *dorycnion*, *strychnon*, *trychnon*, and *solanum* all appear to be non-specific members of the nightshade family. Theophrastus seems to lump several plants under the heading of *strychnos*: one is edible and has berry-like fruit (*Hist.Pl.* 7.15.4) which could possibly be a species of the *Solanum* genus (within the nightshade family) some of which produce fruit that is edible when completely ripe.[33] The second induces sleep (*Hist.Pl.* 7.15.4), while

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32 *bibitur et...ante sectiones punctionesque, ne sentiantur* (*HN* 25.150).

33 Hardin (1974) 143.

the third causes madness (*Hist.Pl.* 9.19.1) (possibly henbane or datura); with a certain dose of the latter, however, a man can be killed (9.11.6). Pliny writes that there are four kinds of *strychnon* (of which *trychnon* is an alternate spelling) (*HN* 21.177-80). Two varieties resemble the leaf of the ivy, but one is a woody shrub, while the third has leaves more like basil (possibly deadly nightshade). A very small amount of this third kind, which some people call *dorycnion* (*HN* 21.179), is said to cause madness, and increasing amounts cause hallucinations, insanity, and death (*HN* 21.178). The fourth kind he calls *halicacabos*, a soporific which kills faster than opium (*HN* 21.180).[34]

According to the *OLD*, henbane was called either *hyoscymus* or *altercum* in antiquity. Datura may have been either *strychnon* or *dorycnion*. Due to the similarities in appearance and effect between these two plants, it is difficult to know whether the ancients were able to distinguish between henbane and datura. They could very easily have been confused. On the whole, *mandragoras* seems to refer to mandrake, except Theophrastus uses *mandragoras* once to refer to what could be *Atropa belladonna* (deadly nightshade); he says its fruit is peculiar in that it is black (*Hist.Pl.* 6.2.9). On the other hand he also writes that the fruit is like grapes or berries (deadly nightshade berries do not grow in clumps like grapes) and is like wine in flavour (*Hist.Pl.* 6.2.9) (the berries of deadly nightshade are particularly toxic).

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34 According to the *OLD*, *halicacabos* refers to various plants; it might be either winter cherry (*Physalis alkekengi*) or sleepy nightshade (*Withania somnifera*). Both plants are poisonous. André, in addition to the above two plants, also suggests *Solanum nigrum* and *Convolvulus oleaefolius* (bindweed). (1985) 115-6.

Since the poisonous members of the nightshade family have similar physiological effects, and some of them are similar physically, there is no need to go to great lengths trying to distinguish the different varieties that appear in the ancient literature. It is sufficient to note that poisonous solanaceous genera grow in the Mediterranean, that they were recognized as both poisonous and medicinal in antiquity, and that some were rightly believed to be deadly.

Colchicum (autumn crocus or meadow saffron) is a member of the lily family, Liliaceae. In the spring, colchicum produces long, glossy leaves which die back before the pale purple to white crocus-like flowers appear in the autumn (this flower is not related to the spring-flowering crocus, a member of the iris family, which it closely resembles).[35] The plant grows from a corm (like a bulb: a short, rounded, underground stem) and prefers damp meadows; it is a native of Europe and Africa and is found throughout the Mediterranean. The whole plant is very poisonous, but the alkaloid colchicine is concentrated in the corm. Colchicine blocks cell division and affects the nervous system. The symptoms of poisoning do not appear until 3-6 hours after ingestion and sometimes may be delayed for 12-48 hours.[36] First there is a burning sensation and feeling of rawness in the mouth and throat, and then the victim has trouble swallowing; nausea, abdominal pain, severe vomiting, and bloody diarrhea follow. Low blood pressure, delirium, anuria, and convulsions may occur. Death is from respiratory failure. Colchicine is

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[35] Descriptions of the autumn crocus are in Polunin (1987) 207-8; Huxley (1977) 144; Turner and Szczawinski (1991) 181-2; and Hardin (1974) 43.

[36] Details of colchicum poisoning are from Martindale (1982) 416-7; Turner and Szczawinski (1992) 181-2; and Hardin (1974) 43.

used medicinally for gout and rheumatism. The taste of the corm is at first sweet, then bitter,[37] and accidental poisonings have occurred both as a result of medicinal use and from eating the corms as if they were onions.[38]

Autumn crocus or meadow saffron was called *ephemeron* or *colchicum* in antiquity. Pliny describes the plant as having the leaves of a lily, but smaller, blue flowers, and a single root useful as a tooth remedy (*HN* 25.170). Milk was the general remedy for poisoning (Pliny *HN* 28.160-1). Dioscorides describes *colchicum* and *ephemeron* as separate plants; *colchicum* kills by choking, and one who consumes it should be given cow's milk as a remedy (*Mat.Med.* 4.84). *Ephemeron* grows in the shade and woods, and the root is said to help toothache (*Mat.Med.* 4.85). Theophrastus writes that death from *ephemeron* poisoning is slow and lingering rather than quick and easy (*Hist.Pl.* 9.16.6); in fact, the symptoms do last a long time because the body is slow to excrete colchicine.[39] Theophrastus also claims that death can be delayed, even up to as long as a year after ingestion, and that there is no hope of curing the latent effects (*Hist.Pl.* 9.16.6). Although the symptoms of colchicine poisoning do not appear immediately but are delayed for several hours and even up to two days, a fact that may have been observed in antiquity, an interval of one year is far-fetched. Slaves were said to take the autumn crocus if they were made angry often (a tantalizing statement left unexplained), but an antidote existed, a well-

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[37] Duke (1985) 137.

[38] Lewis (1977) 60.

[39] Turner and Szczawinski (1991) 182.

known but, unfortunately, unnamed root (Theophr. *Hist.Pl.* 9.16.6). In reality, there is no known antidote for colchicum poisoning — only the symptoms can be treated.

Nicander lists the symptoms of *ephemeron* poisoning; first the victim's mouth itches, then he suffers pain in the belly followed by vomiting and diarrhea (*Alex.* 249-59). Milk is one of several remedies (the others being infusions of a variety of herbs and other plants) (*Alex.* 260-78). Scribonius Largus describes the symptoms as itching in the mouth followed by a burning like that of chewed pepper; next there is pain in the belly and bloody vomiting and diarrhea (*Comp.* 193). The remedies are infusions of herbs and various other plants (*Comp.* 193). Nicander and Scribonius Largus both record symptoms of poisoning that are very like the symptoms of colchicine poisoning described in modern texts, and the choking sensation mentioned by Dioscorides may be referring to another of the symptoms — difficulty in swallowing.

A plant extremely important in antiquity is hellebore. Unfortunately, the identity of this plant remains unclear — it may be either a member of the buttercup family or the lily family. The plant was known in antiquity as both *elleborus* and *veratrum*, and there were two kinds: black and white. The *OLD* defines *elleborus* as a name used for several poisonous plants that were used medicinally, and it identifies black hellebore as *Helleborus orientalis* and assorted species (family Ranunculaceae) and white hellebore as *Veratrum album* (family Liliaceae). Under the entry *veratrum*, the *OLD* says that this is the name for a variety of plants used as both drugs and poisons; the plants are generally believed to be *Helleborus* and *Veratrum* species. Black hellebore is *V.*

atrum/nigrum or *H. cyclophyllus* (or similar species), and white hellebore is *V. album/candidum*.[40] The descriptions of hellebore in the ancient sources are insufficient to securely identify these plants, so both genera will be covered.

In the modern literature, members of the genus *Helleborus* are termed hellebore, while members of the genus *Veratrum* are termed false hellebore. *Helleborus*, a member of the buttercup family, grows up to half a metre in height and bears deeply cut, hand-shaped leaves; the flowers are large, nodding, and range in colour from cream to green to purple.[41] *Helleborus* is a native of Europe, and the species *H. cyclophyllus* and *H. orientalis* grow in the mountains of Greece in woods and bush; depending on the species, hellebore flowers from February to May. The whole plant contains toxic cardioactive glycosides (helleborin and helleborein), similar to those found in foxglove, which slow the heart-rate.[42] The symptoms of poisoning include pain or a scratchy feeling in the mouth or throat, nausea, vomiting, diarrhea, weak heartbeat, dizziness, dilation of the pupils, and disturbed vision. In severe cases, death may result from cardiac arrest. Hellebore has been used in herbal cures as a purgative or abortive and applied externally to get rid of parasites. If inhaled, the dried and powdered rhizome causes sneezing.

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[40] André's categories are similar. *Elleborus niger* is a plant belonging to the genus *Helleborus*, and *elleborus albus* or *candidus* is *Veratrum album*. *Veratrum atrum* or *nigrum* is the modern *Helleborus cyclophyllus*, while *veratrum album* is the modern *Veratrum album.* (1985) 94 & 269. In general then, black hellebore refers to members of the genus *Helleborus*, while white hellebore refers to *Veratrum album*.

[41] Good descriptions of the various species of *Helleborus* are in Polunin (1987) 67; Huxley (1977) 78; and Turner and Szczawinski (1991) 195-6.

[42] Physiological effects and symptoms are from Turner and Szczawinski (1991) 195-6 and Duke (1985) 227.

The false hellebores (*Veratrum* species) belong to the lily family. *Veratrum album* grows up to 1.5 metres high and has distinctive, broad, oval, pleated or ridged leaves; branched stems at the top of the plant carry clusters of small, green-yellow flowers.[43] *V. nigrum* is similar except it has narrow, spiky, black flowers. Both species grow in the mountains and flower from July to August. Both European and North American species are toxic and contain a variety of alkaloids that cause vomiting, reduce blood pressure and slow the heart-rate.[44] The greatest concentration of alkaloids is in the rhizome, but the whole plant is poisonous. Symptoms of poisoning are a burning feeling in the mouth, nausea, salivation, vomiting, diarrhea, abdominal pain, sweating, paralysis, and spasms. Severe poisoning can cause slowing of the pulse, reduced body temperature, shallow breathing, convulsions, and death. *Veratrum* was once used medicinally to lower blood pressure, but due to its relative ineffectiveness, unstable alkaloids, toxicity, and teratogenic effect, it is no longer used. The dried and powdered rhizome has been used in sneezing powders.[45]

The exact identity of the hellebore described in the ancient texts remains a mystery. Even in antiquity there was uncertainty about the physical characteristics of the plant. Theophrastus says that black and white hellebore (*elleboros melas* and *leukos*) merely share the same name, but he adds that some people say that the plants are alike except

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43 A good description of *Veratrum* is in Huxley (1977) 144; North American varieties are discussed in Turner and Szczawinski (1991) 110-2 and Hardin (1974) 46-7.

44 The physiological and poisonous properties of *Veratrum* are from Jaffe (1990) 161 & 164; Martindale (1982) 169-70; Turner and Szczawinski (1991) 110-2; Hardin (1974) 46-7; and Duke (1985) 506-7.

45 Fogh (1983) 175-6.

for the colour of the root (one white and one black), while others say that the leaves are quite different (*Hist.Pl.* 9.10.1). He also says that black hellebore is fatal to horses, oxen, and pigs, but sheep eat white hellebore and are purged by it (*Hist.Pl.* 9.10.2). Both the black and white varieties were said to grow in mountainous regions (Theophr. *Hist.Pl.* 9.10.2-4). The superstitions concerning the gathering of black hellebore are similar to those of the harvesting of mandrake; one should draw a circle around the plant, cut it while facing east and saying prayers, and be on the lookout for eagles (Theophr. *Hist.Pl.* 9.8.8).

Dioscorides' treatment of hellebore is much like that of Theophrastus', but he says that the root has a burning or sharp taste (*Mat.Med.* 4.150-1). The plant purges by causing vomiting, acts as an abortive, and causes sneezing (*Mat.Med.* 4.150-1). Pliny appears to have obtained most of his information on hellebore from Theophrastus, and some of it is similar to Dioscorides' plant, but he adds that black hellebore is a cure for paralysis, madness, dropsy, and gout; a large dose, however, is dangerous (*HN* 25.54). White hellebore was the more fearful of the two drugs; Pliny says that precautions to prevent an excessive reaction should be taken (*HN* 25.56). White hellebore seems to have been a cure-all; Pliny lists numerous disorders that it can alleviate: dizziness, melancholy, madness, gout, dropsy, stomach problems, cough, and fever to mention a few (*HN* 25.60). Both white and black hellebore were the standard remedies for insanity in antiquity (Celsus *Med.* 2.12.1b (*veratrum nigrum*); 2.13.2 & 3.18.17 (*veratrum album*)). Although hellebore and false hellebore belong to distinct families and contain completely different poisonous principles, their physiological effects are similar, a fact which may explain some of the confusion between these plants in antiquity.

One of the most interesting aspects of false hellebore (*Veratrum*) is its teratogenic effect. Pregnant ewes which eat sufficient hellebore on the fourteenth day of gestation give birth to cycloptic lambs; the cyclopia takes the form of two eyes in a single and central orbit, or one eye in the middle of the forehead.[46] Cyclopia, not uncommon in pigs, is also a human birth defect, and it is always fatal.[47] Observation of cycloptic infants and farm animals in pre-literate antiquity, combined with fear and imagination, may have given rise to the myth of a one-eyed race of men. We know that deformed young were born in antiquity, such as two-headed offspring of both humans and animals, and a calf born with its head attached to its leg (Tac. *Ann.* 15.47). Pliny writes of animals with extra limbs and mentions a person born in Egypt who had a pair of sightless eyes in the back of his head (*HN* 11.272). One could speculate that many mythical monsters were based on the observation of a variety of birth defects.

Poppies belong to the family Papaveraceae. The opium poppy, *Papaver somniferum*, was well-known and widely used in antiquity. This tall (up to a metre) annual bears large, showy flowers ranging in colour from white to red or orange with dark centres.[48] The grey-green leaves

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[46] Doull (1980) 589 and photograph in Kingsbury (1965) 77. In one experiment, ewes fed *Veratrum* on the fourteenth day of gestation produced cyclopia and related facial deformities in 100 per cent of their lambs (Keeler (1972) 449).

[47] porcine: Wilson (1977) vol. 1, 28; human: Wilson (1977) vol. 2, 333.

[48] This description of the opium poppy is from Polunin (1987) 75; Turner and Szczawinski (1991) 206-8; Martindale (1982) 1022; and Hardin (1974) 68.

have wavy margins, and the seeds are borne in rounded or oval capsules. Through the whole plant flows a milky juice which oozes out when the stem or the unripe but full-grown seed pod is incised. Opium, the dried exudate, contains a mixture of alkaloids including morphine and codeine and has a distinctive smell and bitter taste. The small, black seeds, commonly used in baking, contain only traces of the alkaloids and are considered harmless. The poppy grows on waste land, alongside roads, and in fields around the Mediterranean.

The soporific and analgesic effects of opium are mainly due to the action of morphine.[49] Eating opium or the unripened seed pods causes stupor, shallow breathing, deep sleep, cold and clammy skin, weak pulse, and cyanosis. Death is from circulatory and respiratory failure. The derivatives of opium (such as morphine and codeine, which occur naturally in opium, and heroin, which is man-made) mimic naturally occurring pain-killers, sedatives and mood enhancers found in the brain (endorphins and encephalins). The fatal dose of opium for the average person can be as little as 300 mg, but tolerance increases with use. It has never been proved that the external use of opium has any analgesic effect, and its use in liniments is a folk remedy. Treatment of opium poisoning consists of emptying the stomach and administration of charcoal and a cathartic, followed by strong coffee as a stimulant.

The effects of opium were well documented in antiquity. Nicander writes that drinking poppy tears (the juice oozes out of the capsules in beads or "tears") causes deep sleep, chilled extremities, sweating, pallor,

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[49] The narcotic and poisonous qualities of opium are from Martindale (1982) 1022; Turner and Szczawinski (1991) 206-8; Hardin (1974) 68; and Duke (1985) 345.

and faint breathing; the precursor of death is either a livid fingernail, distorted nose, or sunken eyes (*Alex.* 433-42). Remedies are hot wine and grape syrup (assuming the victim has not yet fallen into a stupor), or oil; in addition, some attempt should be made to both rouse and warm the victim by slapping his cheeks and rubbing his limbs or immersing him in a hot bath (*Alex.* 443-64). Theophrastus explains the process of gathering the juice, done mainly in the summer, and says that the poppy is peculiar in that the sap is collected from the seed capsule (*Hist.Pl.* 9.8.2); he also mentions that there are several different kinds of poppy all bearing the same name (9.12.3; 9.12.5). Dioscorides describes several kinds of poppy, one being the opium poppy whose leaves and heads soaked in water and drunk are soporific (*Mat.Med* 4.65). He suggests that opium can be used externally for inflammations and that the heads mixed with water and honey can ease coughing (*Mat.Med.* 4.65) (codeine is an ingredient in many modern cough syrups). On the other hand, Dioscorides warns against taking too much poppy as it can kill (*Mat.Med.* 4.65). The best opium was thick and had a distinct odour and bitter taste (*Mat.Med.* 4.65). That there was an opium trade (and probably general drug trade) is indicated by Dioscorides' suggestions for testing the purity of opium; the drug could be adulterated by adding gums or the milky juice of harmless plants (*Mat.Med.* 4.65).[50] He also gives details on how to collect opium; after the morning dew has dried, the heads are incised with a knife, the initial tear is scraped off, and subsequent tears are collected on the following days (*Mat.Med.* 4.65).

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50 According to Allbutt (1921) 350 & 359, there was an open drug and poison trade at markets and also a thriving trade in spices and drugs from the East; actual retail outlets have been identified in Rome and Pompeii.

Scribonius Largus describes the symptoms of opium poisoning as heaviness in the head, coldness and leaden colour in the limbs, cold sweat, hindered breathing, deadened senses, and sleep; treatment is forcing the victim to vomit, administering rose oil and vinegar, rubbing his feet, and preventing him from falling asleep (*Comp*. 180). Pliny's description of opium is similar to Dioscorides': the poppy is a soporific which can be fatal if taken in too great a quantity; the juice is gathered by making incisions in the plant and scraping off the beads; and there are several tests to determine the purity of opium (*HN* 20.198-9 & 203). Pliny's remedies for opium poisoning are salt drunk in vinegar and honeyed wine, and heraclium (some sort of herb) in wine with ashes (*HN* 31.104; 20.178).

Yew is an evergreen, needle-bearing shrub or tree of the family Taxaceae (it is not a true conifer, as it produces neither seed-bearing cones nor resin in the wood or leaves).[51] It produces seeds (it is a gymnosperm), each one of which is surrounded by a soft, red, fleshy cup (an aril, or seed-covering, not a true berry) which is open at the top to expose the seed. The tree is able to grow and flourish in a variety of soils. The entire tree is highly poisonous, including the seed, with the exception of the red "berry" which is edible. The poisonous principle is taxine, a collection of alkaloids which acts on the heart by reducing its conducting ability.[52] Yew also contains taxiphyllin, a cyanogenic

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[51] Descriptions of yew and discussions of its poisonous qualities are in the *Oxf. Enc. Trees of World* (1981) 107-8; Hardin (1974) 42-3; and Turner and Szczawinski (1991) 162-3.

[52] Doull (1980) 587. There is a system of conducting fibres in the heart which initiate its contraction. Yew depresses the activity of these fibres and causes the heart to stop during diastole, when the chambers are filling with blood and before the ventricles contract.

glycoside. Symptoms of poisoning include gastroenteritis, trembling, dilation of the pupils, difficulty in breathing, weakness, collapse, and convulsions. In severe cases there are few distinctive symptoms; convulsions are followed by sudden death (within five minutes) from heart and respiratory failure. Treatment consists of emesis, followed by charcoal, a cathartic and monitoring of symptoms.

The yew tree was clearly recognized in antiquity, probably by its distinctive red arils (called "berries" in antiquity). Pliny correctly identifies it as the only tree of the needle-bearing evergreens that bears berries; he also notes that it has no sap (*HN* 16.50) (yew does not contain resin). He writes that the berries of the male tree are deadly poisonous, especially in Spain (16.50). In fact, the male pollen-producing structures, and the female seeds and "berries" are borne on separate plants, and the berries are only poisonous if the seeds within them are chewed before swallowing. Pliny descends to the realms of folklore when he writes that in Arcadia, sleeping or eating under a yew tree is fatal, and that a yew can be rendered non-toxic by driving a copper nail into its trunk (*HN* 16.51). Nicander warns that the yew is deadly but was under the misconception that plenty of undiluted wine would bring relief (*Alex.* 611-5). Other than this one remedy, the ancient sources do not concern themselves with the treatment of yew poisoning. Theophrastus says that the yew bears round, soft, red fruit which can be eaten by man without harm (*Hist.Pl.* 3.10.2). He also mentions that if beasts of burden eat the needles they will die but that ruminants are immune (*Hist.Pl.* 3.10.2). In fact, cattle are often poisoned from eating yew, and attempts to save them are drastic; the rumen must be opened and the contents removed.[53]

The yew was associated with death in antiquity, possibly solely on the basis of its poisonous qualities. Pliny remarks on its gloomy and dire aspect (*HN* 16.50), and Silius places it in the underworld where it is watered by Cocytus (*Pun.* 13.595-6). The yew of the underworld is a dark, shadowy, shuddering tree according to Seneca (*Her.F.* 689-90), and Tisiphone, one of the Furies, brandishes a blazing yew branch below the earth (Stat.*Theb.* 4. 485-6). Finally, it was generally recommended to keep one's bees away from yew trees (Columella *Rust.* 9.4.3; Verg. *G.* 4.47; *Ecl.* 9.30).

Fungi do not belong to the plant kingdom, but for the sake of convenience I have included them in this chapter. Fungi are organisms that lack chlorophyll and either live off dead matter, form symbiotic relationships with living plants, or act as parasites.[54] The mushrooms we see are actually the spore-producing structures of the organism; a network of minute fibres called the mycelium grows underground and can extend into new areas for many years, whereas the fruit body (the mushroom) lasts for only a matter of days. Since fungi are generally not easy to digest, even edible fungi, if eaten in excess, may make a person feel ill.[55] Some mushrooms contain thermolabile poisons which are destroyed by cooking; they are then safe to eat. The symptoms of

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53 *Oxf. Enc. of Trees* (1981) 107.

54 The following discussion of mushrooms is from Litten (1975) 90-101 and Bresinsky and Besl (1990) 3-4.

55 Since the Roman elite were famous for gorging themselves at banquets, if they happened to eat too many mushrooms, the subsequent feeling of illness might have cast suspicion on edible fungi. On the other hand, gorging would likely make one feel unwell anyway.

poisoning by mushrooms can be brought about by fear of having eaten a poisonous variety; these cases are virtually impossible to distinguish from real poisoning. Old or damaged edible fungi can become poisonous after they have been attacked by bacteria and moulds.

Poisonous fungi can be divided loosely into two groups: fatal and non-fatal. One genus, *Amanita*, contains the deadly mushrooms. One species, *Amanita phalloides* (death cap), is responsible for 90 per cent of the fatal poisonings in western Europe, but *A. verna* (white death cap) and *A. virosa* (destroying angel) are also deadly.[56] These three species have similar physical characteristics; a membranous ring hangs from the stem, the gills are free (they are not attached to the stem), and the base is enveloped in a spherical cup or sheath. The cap of *A. phalloides* is smooth, convex to flat, and olive, yellow-green, or quite pale. The mushroom grows in mixed deciduous forests (particularly under oaks) throughout Europe in the fall. *A. verna* is very like *A. phalloides* except it is white. It is generally restricted to the Mediterranean, and the fruit bodies appear in the spring in chestnut and mixed oak forests. *A. virosa* is also white, but it has a conical cap and grows in mountainous, coniferous forests. All these mushrooms contain amatoxins, cyclopeptides that are unaffected either by cooking or by digestive enzymes.

After eating *Amanita* mushrooms, the symptoms of poisoning do not appear until many hours have elapsed; the usual latent period is 8 to

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56 An excellent description of these three main *Amanita* species is in Bresinsky and Besl (1990) 25-34, from which the following discussion is taken.

12 hours but can be between 6 to 24 hours.[57] Sudden nausea and abdominal pain is followed by vomiting and watery diarrhea and consequent dehydration and loss of electrolytes. This gastrointestinal disturbance lasts for 12 to 24 hours (or even as long as 2 to 4 days) and is followed by a period of apparent improvement. Then, after 1 or 2 days, the victim dies from massive liver and kidney damage (amatoxins attack the nuclei of the cells and disrupt protein synthesis). The lethal dose of *A. phalloides* can be as little as one mushroom; *A. verna* and *A. virosa* contain less poison but still enough so that one or two mushrooms can be fatal. It is said that *A. phalloides* has a pleasant taste.[58] Treatment consists of eliminating the toxin from the gastrointestinal tract, trying to prevent absorption by the liver cells, and maintaining fluid and electrolyte levels. Ignorance or lack of care in identifying mushrooms can lead to confusion between the deadly Amanitas and edible varieties; the white Amanitas and very pale specimens of *A. phalloides* are easily mistaken for edible white fungi.

For the purposes of intentional poisoning, the Amanitas are probably the most devious and most easily administered of all the poisons available in antiquity. Cooked, and sliced or chopped, poisonous mushrooms could be added to an assortment of dishes or mixed in with edible varieties. Mushrooms were considered a luxury in antiquity (Mart. *Ep.* 3.60; 7.78; 12.17), probably because of their brief period of availability and scattered distribution. Romans were fond of fungi but

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[57] The rapid onset of symptoms after eating mushrooms (15 minutes to 2 hours) indicates that non-fatal poisonous fungi have been consumed (Bresinsky and Besl (1990) 18).

[58] Litten (1975) 94.

were also aware of their potential dangers. Pliny says that mushrooms are among the things eaten recklessly (*HN* 22.92), but both Pliny and Celsus observed that some poisonous fungi could be rendered harmless if well-cooked (*HN* 22.99; *Med.* 5.27.12c). The fact that fungi growth occurs after rains, and that the fruit bodies exist for only a matter of days, was recognized in antiquity (*HN* 22.100; 22.95); but mushrooms were also surrounded by folklore. Dioscorides writes that mushrooms were made poisonous by their proximity to rusty nails, rotten rags, or serpents' lairs (*Mat.Med.* 4.83), and Pliny repeats this with the exception of the influence of snakes (*HN* 22.94). Dioscorides also says that fungi are either edible or poisonous, that those collected and stored quickly become spoiled, and that in general mushrooms are hard to digest (*Mat.Med.* 4.83). One remedy for mushroom poisoning was vinegar (Celsus *Med.* 5.27.17; Pliny *HN* 22.99).

Non-fatal poisonous mushrooms were certainly known in antiquity, in addition to methods for rendering some of them edible. The rapid onset of gastrointestinal symptoms from such mushrooms would have been linked to ingestion of the fungi; however, the long latent period of around 10 hours following the ingestion of Amanitas probably disguised the connection between illness and fungus. In addition, another meal could very well be eaten in the intervening hours, thus confusing the association even more. Furthermore, since there was no systematic classification of fungi, and since mushroom lore was probably restricted knowledge, the chances of not always distinguishing between poisonous and edible fungi must have been great.

If Claudius really was poisoned, it is just possible that he was killed by an *Amanita* mushroom.[59] Some ancient sources imply that poison was added to a dish of mushrooms (Suet. *Claud.* 44.2; Tac. *Ann.* 67; Dio Cass. 61.34.2). A simpler explanation is that a poisonous *Amanita* was included in the mushrooms themselves. Since Claudius was extremely fond of mushrooms (Suet. *Claud.* 44.2), generally stuffed himself at the dinner table, and appears to have been prepared to consume any food and drink at any time or place (Suet. *Claud.* 33.1), he would be a better candidate for poisoning than a cautious and finicky eater. The historical accounts of the last days of Claudius are unclear, probably because Suetonius, Tacitus, and Dio — our major sources for his reign — were not alive at the time of Claudius' death; they obtained their information secondhand and well after the fact. Suetonius says that Claudius was poisoned but that when and by whom is unclear (*Claud.* 44.2). After vomiting, Claudius was said to have been given a second dose (*Claud.* 44.3); it is possible that this assumption was actually a reflection of the pattern of *Amanita* poisoning. Gastrointestinal distress, subsequent improvement, then further illness and death may have appeared to be a case of double-dose poisoning.

Tacitus says that Agrippina was responsible for Claudius' death; she sought a poison whose effects would not be immediate but which would defer death in order to conceal her crime (*Ann.* 12.66). She enlisted the help of Locusta (*Ann.* 12.66), a famed poisoner, who may have understood the action of the *Amanita* toxin. Tacitus' version of the events becomes a little confusing at this point; he says that Claudius was

59 Grimm-Samuel has written an interesting article, but she states that Claudius was killed with an *A. phalloides* mushroom. I think that this is only a possibility.

not initially affected by the poison but suffered a movement of his bowels which seemed to help (*Ann.* 12.67). He then adds that Xenophon, the doctor, was called in, and he helped Claudius vomit with the aid of a feather down his throat — a feather that was also said to be tainted with a quick poison (*Ann.* 12.67). Claudius died some time later. It is unlikely that Xenophon could have killed anyone with a poisoned feather; there was no poison in antiquity that could kill if merely touched to the back of the throat with a feather (which could not carry a significant amount of poison), and the immediate reaction of vomiting would remove any toxin that had been swallowed or was still in the region of the throat. The fact that Claudius became ill and died after being visited by the doctor might have appeared to the Romans to be a case of poisoning by physician; it may have been simply the distinctive course of *Amanita* poisoning. Furthermore, the date of Claudius' death, October 13, coincides with the time of fruiting of *A. phalloides*; *A. verna,* however, can produce fruit bodies well into the summer and could be collected, dried, and used at a later date.

The natural world provided a plentiful supply of poisons to the Romans. Although it is unlikely that all of the poisons discussed above were used for deliberate poisoning, there must have been instances of both medicinal overdose and accidental poisoning. Obviously, dosages could not be carefully controlled, nor did the ancients possess sufficient knowledge of poisonous principles and physiological actions to control the effects of potent drugs. The two best poisons for deliberate dispatch would have been aconite and the *Amanita* mushrooms, although colchicum, opium, yew, and members of the nightshade family, given in sufficient quantity could have brought about the desired effect.

CHAPTER III: METALLIC, ANIMAL, AND IMAGINARY POISONS

In antiquity, the botanical world represents the most abundant source of poisons, but mineral and animal sources are also significant. In addition there is the category of fanciful poisons — substances that are not toxic but were believed in antiquity to be deadly. For the most part, lead poisoning was unintentional and probably unrecognized; mercury and arsenic poisoning were rare and more likely due to occupational exposure than to criminal intent. The animal poisons centered around three insects and one amphibian, all of which appear in the Digest as illegal substances,[1] and the imaginary poisons consisted of water, blood, and a sea creature.

Lead is one of the oldest metals known to humankind.[2] Its modern chemical symbol, Pb, comes from the Latin *plumbum* (lead). It is considered a base metal (along with copper and iron) as opposed to a precious or noble metal (such as gold or silver). The dull blue-grey colour of lead is due to the formation of lead carbonate from exposure to air; it actually has a shiny and silvery appearance when cut or cleaned. Lead does not occur naturally in its metallic state, but as a relatively non-toxic ore, lead sulphide, or galena, which looks like a black, shiny stone. If galena is placed in an open fire, it will yield metallic lead. Galena often contains silver as well; if the metallic lead obtained from silver-bearing

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[1] *Dig.* 48.8.3.3 states that among the things which may not be handed over heedlessly, or without good cause, by dealers in cosmetics are a poisonous beetle (*bubrostis/ buprestis*), a poisonous caterpillar (*pityocampa*), the cantharid beetle (*cantharis*), and the salamander (*salamandra*). This section of the Digest also includes the plants hemlock, aconite, and mandrake.

[2] The following discussion of lead is derived from Smith, "What is lead?" (1936); Smith, "Lead in history" (1986) 7-12; and Waldron (1973) 391-2.

galena is heated further, litharge (lead oxide) and silver are produced. Lead is dense, heavy, soft, malleable, and resistant to corrosion, but it has little elasticity or mechanical strength.

Lead beads have been found dating back to 7000-6500 BC in Anatolia.[3] Pre-Roman use of lead was small-scale and restricted to figurines, weights, fishing sinkers, coins, solder, cosmetics (the lead compounds lead sulphide and lead carbonate), pigments, glazes, and glass.[4] Large-scale use of lead came about as the Romans devised ways of supplying the towns and cities of the Empire with water. They made pipes by casting, rolling, and jointing sheets of lead; cisterns and aqueducts were also lined with the metal. Other uses of lead were for roofing, covering ships' keels, anchors, weights, lining bronze cooking pots, utensils, plates, pewter (consisting of half tin and half lead), jewelry, decorative ornaments, medicines, pigments, and lead-lined urns and sarcophagi.

The widespread use of lead inevitably brought exposure to the toxic properties of the metal. Lead has no physiological function in the human body; any lead that is absorbed has the potential to be poisonous. In general, lead forms two kinds of compounds — organic and inorganic. Organic lead compounds contain carbon and hydrogen while inorganic compounds are the salts (compounds consisting of a metal and a non-metal) and oxides of lead, and do not contain carbon — with the exception of the carbonates and cyanides. The lead compounds that were

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[3] Smith, "Lead in history" (1986) 7.

[4] The following discussion is derived from Smith, "Lead in history" (1986) 9-12 and Waldron (1973) 392-3.

significant in antiquity were mainly the inorganic compounds. The principal sites for the absorption of lead are the gastrointestinal tract, the lungs, and the skin.[5] The skin forms a relatively good barrier to inorganic lead compounds, while organic compounds are easily absorbed through the skin and thus can cause poisoning. The main site for lead absorption, however, is the gastrointestinal tract. Adults absorb about 10 per cent of ingested lead, while children absorb 25-50 per cent. Although the lungs absorb about 40 per cent of inhaled lead, and signs of poisoning develop more quickly by this route, the respiratory tract is a secondary site for absorption. Exposure to air-borne lead compounds was certainly less common in antiquity than it was during the past decades of combustion of leaded fuels. A variety of factors promotes absorption of lead from the gut: youth; low dietary calcium; iron deficiency; the consumption of alcohol; the consumption of acids (such as ascorbic or citric); dietary fat (the more fat ingested the more lead absorbed); and an empty stomach or fasting state. A low level of dietary protein decreases the amount of lead absorbed.

Once lead is absorbed, it enters the blood stream and soft tissues, particularly the kidney and liver.[6] Within a month, 90 per cent of the lead is bound in the skeleton where it does no harm to either the ingestor or the bone. The lead remains in the bone unless the skeleton begins to lose calcium, in which case the lead is released into the blood. While lead is in the soft tissues, it exerts a toxic effect on four main organs and

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[5] The following discussion of the absorption of lead is derived from Moore (1979) 243-9; Doull (1980) 415-6; Moore (1986) 54-62; and Arena (1979) 255.

[6] The course of lead in the body and its toxic effects are discussed in Doull (1980) 39, 41, 43, 418-9; Needleman (1985) 78; and Moore (1979) 243.

systems: the central nervous system, peripheral nervous system, kidneys, and blood. The most serious effects, because they are usually irreversible, are on the two nervous systems.

A distinction needs to be made between acute and chronic lead poisoning. Acute lead poisoning refers to a large dose of lead taken over a short period of time, while chronic poisoning occurs from exposure to smaller amounts of lead over a longer period of time. The symptoms of acute poisoning are "intense thirst, an astringent and metallic taste in the mouth, a burning abdominal pain, vomiting (sometimes with milky vomitus), diarrhoea or occasionally constipation, black stools, haemolysis and renal damage with oliguria, shock and coma."[7] The lethal dose for an average person is estimated at 500 mg of absorbed lead; in one case an adult died after swallowing 10 g of lead salt.[8]

The first symptoms of chronic lead poisoning are loss of appetite, constipation, headaches, weakness, and the formation of a blue line on the gums.[9] Anaemia results from the decreased life span of the red blood cells and from the inhibition of the formation of haemoglobin. Mental dullness, irritability, lack of coordination, headache, vomiting, tremor, memory loss, and in severe cases delirium, convulsions, and coma, indicate the involvement of the central nervous system and lead encephalopathy (a change in the nature and structure of the brain). If the victim does not die, he or she may suffer from epilepsy, hydrocephaly,

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[7] Martindale (1982) 935.

[8] Martindale (1982) 935.

[9] The symptoms of chronic lead poisoning are derived from Martindale (1982) 935 and Doull (1980) 418-9.

and idiocy. Damage to the peripheral nervous system results in lead palsy or paralysis, also known as wrist and foot drop. The proper functioning of the kidney can be affected. Lead seems to damage the renal tubules and inhibit the excretion of uric acid, which may result in gout. Lead colic refers to the severe abdominal pain caused by lead poisoning. Lead may cause the uterus to contract, resulting in spontaneous abortion; premature births and stillbirths may also occur.[10] In addition, foetal exposure retards the mental and physical development of children in their first two years of life.[11] Even low or sub-clinical (below the level considered toxic) exposure to lead can affect children; their IQs drop, and they have learning and behavioral problems.[12]

Probably the greatest problem to overcome in dealing with chronic, and especially low-level, lead poisoning is that of diagnosis. The symptoms can be so varied and vague that they can mimic a multitude of different problems.[13] In one case, one third of the patients examined for lead poisoning did not volunteer any complaints or symptoms,[14] and people exposed to low levels of lead are unlikely to exhibit the revealing symptoms of anaemia, wrist drop, and colic.[15] The early symptoms of lead poisoning may resemble a virus or simple stress, and the variety and

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[10] Smith, "Lead in history" (1986) 23.

[11] "Lead poisoning from ceramics" (1988) 1358.

[12] Arena (1979) 257; Doull (1980) 418; "Lead poisoning from ceramics" (1988) 1358.

[13] Nriagu *Lead* (1983) 379-80; Arena (1979) 257; Pagliuca (1990) 830; "Lead poisoning from ceramics" (1988) 1358.

[14] Pagliuca (1990) 830.

[15] Keogh (1984) 1945.

combinations of signs involving the four target systems can resemble the following problems: appendicitis, duodenal or gastric ulcer, renal colic (severe pain caused by obstruction of the ureter by a kidney stone or blood clot), gastroenteritis, porphyria (a metabolic disorder characterized by recurring abdominal pain, vomiting, constipation, and varying degrees of paralysis), heat exhaustion, intestinal parasites, infection, malnutrition, intracranial pressure from tumours or infection of the meninges (the membranes covering the spinal cord and brain), and the blue gum line can be caused by gingivitis or poor dental hygiene.[16] One investigator described the symptoms of low-level lead poisoning to two general practitioners and asked them to suggest possible diagnoses. One doctor recommended rest and tranquilizers, while the other guessed malaria.[17] Another example of the insidiousness of lead poisoning is that of a couple who bought lead-glazed pottery mugs in Italy and began to drink coffee from them once they returned home to Seattle. The husband began to suffer from insomnia, weight loss, and pain in the wrists, and he underwent two unnecessary and ineffective carpal-tunnel (wrist) operations to correct the problem. The wife was diagnosed with porphyria. Only after the couple undertook their own research and insisted on being tested for lead poisoning was the mystery solved.[18]

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[16] Nriagu, *Lead* (1983) 379, citing Repko and Corum, p. 141 in *CRC Crit. Rev. Toxicol.* 6 (1979) 135-87.

[17] Nriagu, *Lead* (1983) 379. Of course, since the doctors had no opportunity of making a physical examination, they were at a disadvantage. Described symptoms without the benefit of examination and questioning by a physician could naturally lead to mis-diagnosis.

[18] "Lead poisoning from ceramics" (1988) 1358.

Just how much the Romans were affected by lead in their environment has been hotly debated. The theory that lead poisoning was the major factor in the fall of the Roman Empire[19] is an extreme view and cannot be taken seriously, but it does seem reasonable that the Romans consumed more lead in their diets than what is now considered allowable. Given the amount of lead used in Roman water-delivery systems, it has been suggested that the Roman water supply was severely contaminated. In fact, the amount of lead that leaches into a water supply depends on the type of water it holds. Soft or acidic water is particularly plumbosolvent or corrosive to lead and causes enough lead to enter a water supply to cause poisoning.[20] Hard water, which has a higher mineral content than soft, leaves a deposit of calcium carbonate on lead which protects the water supply from lead leaching from the pipes. The modern water supply at Rome is hard, and there are limestone deposits on the old aqueducts; however, ancient Rome's water came from a variety of sources, and it has been estimated that one quarter of these were soft and mildly acidic.[21] But a factor in Rome's favour was that the water flowed continuously; flowing water spends less time in contact with its pipe and thus dissolves less lead than standing water.[22] Nevertheless, Vitruvius made some insightful comments regarding lead pipes. He wrote that water is much more healthful from earthenware pipes than from

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19 Nriagu, *Lead* (1983) viii & 415.

20 Smith, "What is lead?" (1986) 5; Nriagu, *Lead* (1983) 321; Waldron (1983) 181; Arena (1979) 256.

21 Nriagu, *Lead* (1983) 321.

22 Needleman (1985) 74.

lead pipes because lead produces cerussa (lead carbonate or white lead), an acknowledged poison (Vitr. 8.6.10). He added that in order to be healthful, as little water as possible ought to be brought through lead piping and that the flavour of water in earthenware pipes is better (Vitr. 8.6.11). Vitruvius appears to have been aware of the potential danger of lead poisoning.

Romans probably ingested most of their lead in wine and food.[23] Must (unfermented or partially fermented grape juice) was boiled down to form *sapa*, *defrutum*, or *caroenum*, which was added to wine as a sweetener, preservative, and flavouring and colouring agent.[24] Columella states that the vessels used for the long boiling process ought to be lead rather than bronze since bronze will spoil the flavour.[25] He adds that the more the must is boiled down the better and thicker it becomes (*Rust.* 12.20.3), and as a guideline he suggests that the must be reduced by one quarter to one third of its original volume; must reduced to one half its volume, however, would make a better and thicker *sapa* (*Rust.* 12.19.1). Pliny confirms the preference for lead over copper vessels for boiling *sapa* and *defrutum* (*HN* 14.136). *Sapa* was added to poor wines as a sweetener and flavour-enhancer (Pliny *HN* 14.121). As a preservative it extended the shelf-life of any wine due to the ability of lead to inhibit enzyme activity.[26]

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23 Analysis of skeletal remains from Roman Britain shows concentrations of lead similar to or greater than concentrations expected in modern, industrial societies. The study concludes that, in this case, the lead must have come from food and drink. Waldron (1983) 172-82.

24 Nriagu, *Lead* (1983) 341 and "Sat gout" (1983) 660.

25 *Ipsa autem vasa, quibus sapa aut defrutum coquitur, plumbea potius quam aenea esse debent. Nam in coctura aeruginem remittunt aenea, et medicaminis saporem vitiant* (*Rust* 12.20.1-1).

26 Waldron (1973) 393.

During cold weather wine was often warmed, and lead kettles have been discovered at Pompeii.[27] The warming action and contact with a lead surface would increase the lead content of the wine further.

Lead in food probably came from a variety of sources. In its capacity as a preservative, *sapa* was added to preserved fruit to keep it from fermenting.[28] Although honey was a common sweetening agent, grape syrup was a less expensive alternative.[29] According to Nriagu, either *defrutum* or *caroenum* is called for in about 20 per cent of the recipes in Apicius' cookbook.[30] The grape syrup was used in sauces for meat, fowl, and fish, in broths and soups, as a seasoning for vegetables, and in pastries. Another source of lead was cooking pots and utensils. Pliny says that vessels lined with *stagnum* (an alloy of silver and lead) impart a more agreeable taste to their contents (*HN* 34.160). The Roman bronze used for utensils contained some lead; the bronze of Campania, esteemed for utensils, contained 5 to 8 per cent lead and pot bronze contained 1.5 to 2 per cent lead (Pliny *HN* 34.95-6 & 98). The Romans also used pewter, and examples of pewter frying-pans, bowls, and pots have been found in a variety of Roman sites, including Pompeii.[31] Roman pewter was 50 per cent tin and 50 per cent lead.[32] Yet another potential source of lead in the diet was the venerable olive. Columella gives

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27 Nriagu, *Lead* (1983) 347.

28 Waldron (1973) 393; Nriagu, *Lead* (1983) 336.

29 Needleman (1985) 74.

30 Nriagu, *Lead* (1983) 334-5.

31 Nriagu, *Lead* (1983) 332.

32 Smith, "Lead in history" (1986) 12.

instructions on how to make beeswax water: water in which beeswax has been soaked is boiled in a leaden vessel to the consistency of *sapa*. The resulting liquid can be used instead of honey-water, which was drunk, or in place of boiled-down must for preserving olives (*Rust.* 12.11.1). One more potential source of lead poisoning is pottery decorated with lead-based glazes, which appeared in the first century B.C., but the everyday earthenware of the Roman household was normally left unglazed.[33]

Investigators have tried to produce *sapa* and make wine using ancient recipes and instructions. The resulting syrup has yielded from 240 to 1000 mg of lead per litre, and the wines have yielded 15 to 30 mg of lead per litre.[34] One rather alarming, although probably infrequent, use of *sapa* was as an antidote (Pliny *HN* 23.62). In this case the medicine may very well have been worse than the malady. Attempts to estimate the daily lead intake of the Romans have resulted in a wide range of figures, all of which are the product of guesswork. Nriagu suggests a range of lead absorbed daily from all sources running from 15 μg for slaves to 250 μg for aristocrats.[35] According to Nriagu, the slaves were at low risk due to their infrequent access to the best wine and delicacies; as drink, the best they could hope for was afterwine, grape skins soaked in water.[36] Needleman and Needleman come up with a figure of 270 μg of absorbed lead or about 3.4 mg of dietary lead daily,

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33 Needleman (1985) 76; Singer (1957) 273.

34 Needleman (1985) 75; Nriagu, "Saturnine gout" (1983) 660, 661.

35 Nriagu, *Lead* (1983) 400, table 6.3.

36 Nriagu, *Lead* (1983) 401, n. k; Cato *Agr*. 57. Cato refers only to the drink of field hands and does not mention urban slaves. Nriagu's figures include lead from all sources (air, food, water, etc.), but he fails to explain how he determined these values.

assuming an absorption rate of 8 per cent.[37] The current maximum allowable concentration of lead in drinking water is 50 µg per litre, although the most recent studies suggest that it should be reduced to 20 µg per litre or less,[38] and the maximum weekly intake for adults is 3 mg.[39]

Although Roman dietary lead intake appears to have been greater than what is now considered safe (and our own maximum tolerable levels are dropping due to increasing evidence of the effects of sub-clinical exposure to lead), it is difficult to know exactly how much lead was consumed on a day-to-day basis. For example, although *sapa* was used to improve inferior wine, it was probably not used in the best wines. In addition, Pliny mentions several other substances used as additives to wine: gypsum in Africa, potter's earth, marble dust, or salt in Greece, and pitch in Italy (*HN* 14.120). The daily diet of most Romans probably did not contain all the sapa-enhanced delicacies imagined by Nriagu;[40] it is possible that these were reserved for special occasions and for those people who could afford them. If the Romans did consume as much lead as is estimated, one would expect the ancient literature to be full of descriptions of mass lead poisoning, or at least some of the more peculiar and obvious symptoms such as wrist-drop. In consequence, it is likely that Roman exposure to lead was lower than what has been estimated.

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37 Needleman (1985) 79.

38 Graziano and Blum (1991) 142.

39 Martindale (1982) 935

40 The average diet in Rome seemed to consist mainly of grains and vegetables with an increasing interest in more diverse foods from the Republic to the Empire. Forbes (1965) v. 3, p. 102-3.

Nicander does describe the effects of two different lead compounds, but both descriptions suggest acute rather than chronic poisoning. He says that litharge (lead monoxide) produces violent and painful colic, a reduction or cessation of urine, swollen limbs, and leaden skin (*Alex.* 594-600). The effects of psimithium (white lead) are an astringent froth in the mouth, dry throat, dry retching, fatigue, chilling, and hallucinations (*Alex.* 74-86). Nicander appears to have got some of the symptoms right; according to Martindale, the symptoms of poisoning by lead compounds are the same as those for metallic lead: abdominal pain (Nicander's colic), oliguria, an astringent taste in the mouth (astringent froth), intense thirst (possibly Nicander's dry throat), and shock (the fatigue and chilling).[41]

Scribonius Largus describes similar symptoms. He implies that litharge causes abdominal colic and oliguria; he also adds that the victim turns a leaden colour (*Comp.* 183). The consumption of white lead causes a whitening of the tongue and gums, nausea, vomiting, blurred vision, dizziness, and stoppage of the breath (possibly coma) (*Comp.* 184). Pliny identifies both white lead and litharge as being lethal poisons (*HN* 34.175-6). Celsus prescribes a remedy for white lead poisoning: mallow or acorn juice rubbed in wine (*Med.* 5.27.12b). Pliny's suggested remedies are ass's milk (*HN* 28.129), mallows (20.223), oil of oenanthe (23.80), and wild figs taken in drink (23.128).

The absence of descriptions of chronic lead poisoning from the ancient literature is not surprising, since the symptoms of low-level and chronic lead poisoning are vague, non-specific, and notoriously difficult

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[41] Martindale (1982) 935.

to diagnose. Even in relatively modern times chronic lead poisoning has been diagnosed as a disease; in France epidemics characterized by pallor, mind and vision disturbance, insomnia, abdominal pain, anorexia, nausea, vomiting, and paralysis in 1616 and 1754 were labeled colic,[42] and chronic lead poisoning continued to be diagnosed as endemic colic until the mid-nineteenth century.[43] The diagnosis of lead poisoning continues to be elusive as we approach the 21st century.

There is little mention in the ancient sources of occupational exposure to lead. Since many of the workers in lead were slaves, it is unlikely that physicians would be called in to examine and treat them, and since the symptoms can be vague, a slave might have been assumed to be shirking duties with complaints of stomach pain, insomnia, and anorexia. The miners themselves were at low risk for lead poisoning since the ore, galena, is relatively non-toxic.[44] Vitruvius does describe a condition that sounds like occupational exposure; he describes the pallor of workers in lead and suggests that the fumes from lead casting get into the limbs of the body and take away the properties of the blood (8.6.11). He appears to have observed lead-induced anaemia. Pliny describes a method of preparing lead for medicinal purposes, which involves melting lead between two layers of sulphur. He suggests that while the lead is melting, the breathing passages should be covered to avoid the harmful and unhealthy fumes of the lead furnace (*HN* 34.167). Of course it is

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42 Smith, "Lead in history" (1986) 19.

43 Stevenson (1959) 15.

44 Waldron (1973) 394; Smith, "Lead in history" (1986) 17.

possible that it was actually the sharp smell of sulphur that Pliny was referring to.

Some of the cosmetics in ancient Rome were compounds of lead. Lead carbonate (white lead) was used as a face powder, and red lead was used as rouge.[45] Lead sulphide or galena was used to accentuate the eyes, but soot was also popular.[46] The absorption of inorganic lead through the skin is relatively insignificant;[47] opportunities for lead absorption would come about from lead on the face and lips working its way into the mouth, hand contact with the cosmetic followed by mouth contact, or inhalation of the various powders during preparation and application. Absorption could also take place through the conjunctival sac.[48]

Lead can be absorbed through cut or abraded skin, and some of the medicinal uses of lead in antiquity were probably an additional source of lead. Celsus gives a recipe for a wound plaster, and the most significant ingredients present in the greatest proportions are white lead, litharge, and lead slag (*Med.* 5.19.26). He also mentions numerous other plasters containing lead; just two others are one for bleeding wounds (5.19.2), and another for wounds considered not too serious (5.19.23). Pliny writes that lead by itself is used for removing scars (*HN* 34.166). He also relates an innocuous, if uncomfortable, use of lead; lead plates, due to their chilly nature, applied to the loins and kidneys, will inhibit

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[45] Needleman (1985) 76; Forbes (1965) v. 3, p. 41.

[46] Forbes (1965) v. 3, p. 42.

[47] Doull (1980) 415; Needleman (1985) 76; Martindale (1982) 936.

[48] Nir (1992) 417; Nriagu, *Lead* (1983) 319.

venereal impulses and spontaneous emissions from libidinous dreams (34.166).

It is unlikely that the ancients associated the violent reactions of acute poisoning from lead compounds with metallic lead or that they regarded the substances, which have different properties and appearances, as containing the same poisonous principle. Since the Romans used so much lead, both in grand projects and in daily life, it is unlikely that they could have remained free from the effects of lead; they probably suffered, unawares, from low-level, chronic lead poisoning.

Even though we are more aware than the ancient Romans of the dangers of exposure to lead, the problem continues, and the statistics are relevant to our understanding of conditions in antiquity. Increasing knowledge and new technology indicate that our current allowable limits on exposure to lead may be unsafe. Although modern water delivery systems no longer use lead pipes, contamination from lead still occurs from the following sources: the lead solder used to join copper piping can leach lead into the water supply; chlorination increases the acidity of water and thus its plumbosolvency; lead solder in kettles; standing water absorbs more lead than flowing water; and the greater the temperature of the water, the more lead is dissolved into it.[49] Although high lead levels have been found relatively recently in households in the United States (as high as 1 mg/l for standing water and .2 mg/l for running water), under normal circumstances lead levels can be reduced to negligible levels by prolonged flushing.[50] It is doubly important to try to reduce the lead in

49 Moore (1986) 150-1.

50 Elwood (1986) 140.

drinking water, since a greater amount of lead is absorbed by the body from water than from food (40-50 per cent in one experiment); in addition, lead in cooking water is readily absorbed by food and vegetables.[51]

Lead poisoning can arise from unexpected sources. An entire family suffered acute lead poisoning after drinking orange juice kept in a glazed ceramic jug from Mexico, and a woman was diagnosed with lead encephalopathy after drinking cider for four months from a partially lead-glazed mug.[52] The lead concentration in port after it had been stored in a crystal decanter for four months was 70 times the allowable limit, and brandy stored for more than 5 years in decanters was found to contain more than 400 times the safe limit.[53] White wine poured into crystal glasses showed an increase in lead concentration after a few minutes and had surpassed the allowable limit in less than one hour.[54] Although the Romans did not use leaded glass, these examples illustrate the ease with which lead may be transferred from vessels or containers to food and drink. As seen in the chapter on plant poisons, practitioners of traditional medicine can be sources of poisoning. In two separate cases two men sought the advice of herbalists, one for impotence, the other for psoriasis. Both received and ingested powders containing 49 per cent

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51 Elwood (1986) 141.

52 Jug: "Lead poisoning," (1988) 1358. Mug: Zuckerman (1991) 550.
A bright red glaze usually contains the most lead, and lead-glazed dishware is considered to be the largest single source of dietary lead in the United States. ("Dishware Hazard" *Globe and Mail* Jan. 23, 1993, p. D8)

53 Graziano and Blum (1991) 141-2.

54 Graziano and Blum (1991) 142.

and 84 per cent lead respectively; both suffered from the symptoms of lead poisoning and were diagnosed and treated by conventional physicians.[55]

Mercury was another metal with the potential to cause poisoning in antiquity. It was not used on such a large scale as lead, so exposure to its noxious effects was far less frequent. In antiquity, mercury existed in its metallic state as *argentum vivum* or *hydrargyrus* and in a compound (mercuric sulphide) called cinnabar or *minium. Hydrargyrus* actually refers to mercury obtained from cinnabar and was not thought to be 'real' quicksilver (*argentum vivum*) in antiquity.[56] Mercury is a curious metal in that it exists in a liquid state and is mobile; it easily divides and coalesces into shiny, silvery spheres. Acute poisoning from mercury occurs from the ingestion of soluble mercury compounds,[57] and, since cinnabar is highly insoluble in the human gastrointestinal tract,[58] its ingestion would not have presented a hazard in antiquity. Chronic poisoning occurs from the inhalation of mercury vapour or the dust of mercury compounds, absorption of mercury through intact skin, and ingestion of weakly soluble mercury compounds.[59] Swallowing elemental mercury is relatively harmless since absorption by diffusion through the gastrointestinal tract is limited by the relatively large size of the mercury

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55 Impotence: Dolan (1991) 630-1. Psoriasis: Smitherman (1991) 795-8.

56 *OLD* (1982) 810; Pliny *HN* 33.123.

57 Martindale (1982) 937.

58 Fischbein (1992) 10.

59 Martindale (1982) 937; *Merck Index* (1989) 927.

particles.[60] The effects of chronic mercury poisoning are muscular tremors, jerky gait, inflammation of the mouth and gums, kidney and liver damage, spasms of the extremities, depression, change of personality, deterioration of mental capabilities, irritability; excessive salivation and loose teeth are indicative of advanced poisoning.[61]

In antiquity, elemental mercury was used in the gilding process (Pliny *HN* 33.64,100; Vitr. 7.8.4), and the mercury compound cinnabar was used as a pigment (Pliny *HN* 33.111-2; 117; 120-1). There was surely some occupational poisoning from handling mercury or inhaling its vapour in antiquity, but it most likely affected the small portion of the population that worked with mercury. Dioscorides and Pliny describe the safety measures taken by workers roasting cinnabar and involved in the process of polishing surfaces painted with the pigment; they tied protective coverings over their faces to prevent inhalation of the dangerous vapour and dust (*MatMed* 5.09; *HN* 33.122). There is no mention of any form of protection for those mining cinnabar and exposed to droplets of mercury (Vitr. 7.8.1), nor for those heating and drying cinnabar, an action which produces mercury (Vitr. 7.8.2).

Swallowing mercury was believed by some authorities to be dangerous. Paulus writes that the ingestion of mercury brings on the same symptoms as litharge, that is, heaviness in the gastrointestinal tract, damage to the intestines, retention of urine, swelling of the body, and a leaden discolouration of the skin (5.63 & 61). Pliny lists many remedies for mercury (*argentum vivum*) poisoning such as parsnip (*HN*

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60 Doull (1980) 33.

61 *Merck Index* (1989) 927; Martindale (1982) 937.

20.35), nettle (22.31), undiluted wine (23.43), ass's milk (28.129), lard (28.158), and the dung of wild doves (29.105). As previously seen, elemental mercury is relatively non-toxic; therefore, no remedy is required, but any of these cures would produce the illusion of effectiveness against perceived poisoning. The Arab practitioners appear to have had a more secure grasp of the properties of mercury; they write that pure or metallic mercury causes no harm when drunk.[62]

Although arsenic compounds were known and used in antiquity, arsenic in its elemental form was not known until the mediaeval period, and the earliest recorded instructions for its reduction to elemental form were provided by Paracelsus in the early 16th century.[63] The two arsenic compounds used in antiquity were realgar (*sandaraca*; arsenic disulphide) and orpiment (*auripigmentum*; arsenic trisulphide). Realgar was used as a red pigment, and orpiment, a bright yellow colour, was used both as a pigment (Pliny *HN* 35.30; 35.49) and as one of many depilatories (Pliny *HN* 34.178). Both realgar and orpiment have caustic properties and were used medicinally as corrosives (Celsus *Med.* 5.7.1). Pliny suggests that realgar can also be used in eye medicines, mixed with turpentine and eaten with food to cure coughs, and taken with honey to improve the voice (*HN* 34.177). Pliny does not mention any poisonous characteristics of arsenic compounds, but Paulus includes realgar and orpiment in his list of poisons. He says that if the substances are drunk they cause severe gnawing pains in the belly and intestines (Paul. Aeg. 5.60). Acute arsenic poisoning in fact does do severe damage to the

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62 Adams in Paulus (1844) 238.

63 *Merck Index* (1989) 126.

gastrointestinal tract.[64] In general realgar and orpiment appear in the literature as therapeutic agents and pigments rather than as poisons.

There is limited evidence concerning oral poisons extracted from animals in antiquity; animals were obviously not considered a bountiful source of poisons, although some substances derived from them were used medicinally. One exception is the cantharid or blister beetle. This insect is called the blister beetle for good reason — it contains the powerful irritant cantharidin, a substance found in the insect's elytra, the hard and protective wing covers.[65] In antiquity, *cantharis* seems to have referred to beetles belonging to the family Meloidae and particularly to the genera *Lytta* and *Mylabris*.[66] The most important of these beetles in mediaeval and modern times has been *Lytta vesicatoria* or Spanish fly, which is a metallic green insect that eats the leaves of the ash, the elder, and other plants.[67] The species used more often in antiquity were the members of the genus *Mylabris*, distinguished by the black and yellow bands stretching across their wings.[68] The life cycle of the Meloidae is complicated and was probably unknown to the ancients; in their larval stage the beetles act as parasites in the nests of solitary bees.[69]

Popularly known as Spanish fly, the Meloidae now have a reputation as an aphrodisiac. When ingested, cantharidin irritates the

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64 Harte (1991) 220.

65 Davies (1986) 92.

66 Beavis (1988) 168.

67 Beavis (1988) 168.

68 Beavis (1988) 168.

69 Beavis (1988) 169.

genito-urinary tract and can cause priapism.[70] In antiquity, however, there is no evidence for its use as a sexual stimulant, an application not seen until the sixteenth century.[71] Swallowing the dried and crushed beetle produces the following symptoms: burning pain in the throat and stomach; swelling and blistering of the tongue, accompanied by difficulty in swallowing; nausea; vomiting; colic; bloody diarrhoea; tenesmus; burning pain in the back, bladder, and urethra; frequent but painful, slow, and bloody urination; thirst; chilling; temporary loss of consciousness; depression of the heart and respiration and eventual circulatory failure; possible delirium, convulsions, and coma.[72] Post-mortem findings show necrosis of the oesophagus and stomach linings, excessive accumulation of blood in the genito-urinary tract, and bleeding in the ureters and bladder.[73] For the average man, a lethal dose of the dried beetle can be as little as 1.5 to 3 g, and toxic symptoms can be elicited with 0.6 g.[74]

Cantharides were once used medicinally on the skin as a blistering agent, irritant, and rubefacient.[75] Since cantharides are highly toxic if absorbed through the skin or mucous membranes, their use in medicine, both internally and externally, has been discouraged.[76] The modern

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70 *Ox. Comp. Med.* (1986) 168; Arena (1979) 405.

71 Davies (1986) 93.

72 Martindale (1982) 1689; Arena (1979) 405

73 Arena (1979) 405.

74 Martindale (1982) 1689.

75 Martindale (1982) 1689; *Oxf. Comp. Med.* (1986) 168.

76 *Merck Index* (1989) 1754; Martindale (1982) 1689.

treatment for cantharid poisoning includes emesis, activated charcoal, demulcent drinks (which are adsorbed and form a protective coat) for the pain in the mouth and throat, heat applied to the abdomen, and circulatory support by intravenous plasma or electrolytes; alcohol, fat, or oils must not be given as they promote the absorption of the toxin.[77]

In the ancient sources, the cantharid beetle seems to come in several varieties, which is not surprising since all the members of the Meloidae are producers of cantharidin. Pliny and Dioscorides say that the most powerful beetles have yellow bands across their wings and are plump (*HN* 29.94; *MatMed* 2.65). Less potent is the variety that is small, broad, and hairy (*HN* 29.94), and both authors agree that the least useful is of one colour (*HN* 29.94; *MatMed* 2.65). These last two varieties may be members of the family Cantharidae and have been tentatively identified as soldier-beetles; soldier-beetles are similar to the Meloidae in form and in the softness of their bodies, but their elytra are covered with small hairs.[78] Dioscorides and Pliny both discuss the preparation and storage of cantharides. The beetles are put in an earthenware pot, the mouth of which is then covered with a cloth; the pot is hung over boiling vinegar until the vapour has killed the beetles (*MatMed* 2.65; *HN* 29.95). Pliny then says that the beetles are stored, while Dioscorides adds the detail that the insects are strung together with a thread before storage (*MatMed* 2.65; *HN* 29.95).

Pliny clearly did not understand the life cycle of the cantharid beetle; he writes that the beetle is produced from the little worm inside

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77 Martindale (1982) 1689; Arena (1979) 405.

78 Beavis (1988) 169.

the sponge-like substance that is found on the wild rose (*HN* 29.94). This 'sponge' is a gall and has been identified as being produced by a certain gall wasp; it is the wasp's offspring that grow within the gall.[79] Alternatively, he proposes generation of the cantharid beetle from grubs on figs, pears, and pines (*HN* 11.118). The association of cantharides with roses may merely indicate confusion between meloids and rose chafers, which have a green colour similar to that of *Lytta*.[80]

The cantharid beetle was certainly recognised as poisonous in antiquity, yet it was also used medicinally. There was debate about which part of the beetle contained the poison; some people thought the feet and the head, but others disagreed (Pliny *HN* 29.94). Everyone agreed, however, that the wings 'helped' (Pliny *HN* 29.94). This statement is problematic in that it is unclear whether they thought that the wings decreased the effect or increased the potency of the poison. Pliny writes that cantharides taken in drink is a poison and causes excruciating pain in the bladder (*HN* 29.93). Nicander records the symptoms of blister beetle poisoning as a biting sensation from the mouth to the belly, pain in the belly and bladder, and delusions; the beetle itself smells of pitch and tastes like juniper berries (*Alex.* 115-27). Scribonius Largus describes the blister beetle as having the taste and smell of cedar pitch; the effects of consuming the poison are pain and smarting in the stomach but especially in the bladder, bloody urine, and loss of consciousness or life (*Comp.* 189). Paulus describes the taste of pitch, the pain from the mouth to the bladder, problems in urination, blood in the

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79 Beavis (1988) 216-7.

80 Beavis (1988) 170.

urine and faeces, nausea, dizziness, and delirium (Paul. Aeg. 5.30). The remedies for cantharidin poisoning are numerous. Pliny suggests must (*HN* 23.29), sapa (23.62), oil of oenanthe (23.80), cow's milk (28.160), goat's milk (28.161), and the broth of ram's flesh (29.105). Nicander recommends pennyroyal in water, meat broth, emesis, milk enema, a draught of milk, grape syrup, and Samian earth (*Alex.* 128-56). Scribonius Largus says that victims are helped, after they have vomited, with raisin wine drunk with oil or milk; in addition he suggests a variety of seeds, fatty meat broth, porridge cooked with plenty of goose fat, and Samian earth (*Comp.* 189). In fact, any of the above remedies containing oil, fat, or alcohol would promote absorption of cantharidin and increase the severity of its toxic effects.

The medicinal uses of the blister beetle were both external and internal. Celsus includes it in a list of caustics (*Med* 5.8.1), and in recipes for a strong corrosive (5.22.2c) and for a treatment for papules (5.28.18b). Pliny says that the general property of cantharides is to cauterize flesh and form scabs (*HN* 29.95). He describes its use against mange (*HN* 29.110) and for removing abscesses (30.75) and warts (30.81), but he warns that care must be taken in the application of cantharis in order to avoid causing deep sores (29.110). Cantharides have actually been used in modern medicine for wart removal.[81] Pliny also includes uses such as smoothing rough nails (30.111) and for psoriasis (30.120). The internal use of cantharides was hazardous; a man who suffered from a skin problem called lichen died after swallowing a treatment consisting of blister beetle in drink (Pliny *HN* 29.93). Blister

[81] Martindale (1982) 1689.

beetle was also said to bring on menstruation and urination (Pliny *HN* 29.95), but this procedure must have been both painful and dangerous.

In addition to being included in the list of forbidden substances in the Digest (48.8.3.3), Pliny says that the sale of cantharides at an auction for 60,000 sesterces was the reason for a charge against Cato Uticensis (*HN* 29.96). Cantharides was also a means of suicide (Cic. *ad Fam* 9.21.3), and in one case revenge. To avenge his brutalized country, a physician prescribed an ointment containing *cantharis* for an eye disease afflicting the tribune Decius (c. 280 BC); he was blinded as a result (Diod. Sic. 22.1).

A beetle pharmacologically similar to but morphologically different from the blister beetle is the *buprestis*. The name means "cow-inflater", and cattle were said to blow up and burst if they accidentally ate the insect while grazing.[82] Attempts at modern identification of *buprestis* suggest that it is a member of the same family as the cantharid beetles (Meloidae) but of the genus *Meloe*, the members of which carry a significant amount of cantharidin in their elytra.[83] These beetles are commonly known as oil-beetles because they exude an oily liquid as a defence mechanism.[84] They have long legs, soft bodies, and short elytra which expose the abdomen; they range in colour from blue-black to metallic green, and they come in a variety of sizes.[85] Their natural habitat is cow pastures, and their life cycle is complex and similar to that of the

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[82] Davies (1986) 91; Pliny *HN* 30.30

[83] Beavis (1988) 173-4; Davies (1986) 91.

[84] Beavis (1988) 174.

[85] Beavis (1988) 173-4.

cantharid beetle in that the larval stage acts as a parasite in the nests of solitary bees.[86]

Nicander does not describe *buprestis*, but Dioscorides says that it is a kind of *cantharis* (*MatMed* 2.65). Pliny states that the *buprestis* is rare in Italy and very similar to a long-legged beetle or scarab (*HN* 30.30). *Buprestis* was a recognized poison in antiquity: it appears in the Digest on the list of poisons (48.8.3.3); its effects are described by Nicander, Scribonius Largus, and Paulus; and numerous remedies to combat its poison were recorded. *Buprestis* also had medicinal uses; it was prepared in a similar fashion to cantharides and had the same property — to cauterize flesh and form scabs (Pliny *HN* 29.95). Nicander, Paulus, and Scribonius Largus give similar accounts of the characteristics and effects of swallowing buprestis: the taste is like soda and is followed by pain in the belly and oliguria; the abdomen swells and the skin tightens (Nic. *Alex.* 335-46; Paul. Aeg. 5.31; *Larg*. Comp. 190). Dioscorides mentions its use as an emmenagogue and diuretic, but he says that its wings and feet are a remedy for cantharides poisoning (*MatMed* 2.66). Such a remedy, however, would surely add fuel to the fire of cantharid poisoning. Other remedies for *buprestis* poisoning were similar to those for the poison of cantharides: must (Pliny *HN* 23.30), sapa (*HN* 23.62), oils (*HN* 23.80; 23.87), milk (*HN* 28.74; 29.105), and soda (*HN* 31.119). Any oily or fatty remedies would, as in the case of the blister beetle, increase the absorption of cantharidin, and soda, being alkaline, should also not be given.[87]

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[86] Beavis (1988) 174.

[87] Arena (1979) 405.

The last of the insects believed poisonous in antiquity is the *pityocampa*, a kind of caterpillar. In the ancient texts, *pityocampa*, *buprestis*, and *cantharis* are generally mentioned as a group since all were believed to have similar properties. *Pityocampa* is included in the Digest's list of poisons, along with *buprestis* and *cantharis*. The identity of the *pityocampa* is thought to be *Thaumetopoea pityocampa* or the pine processionary caterpillar — the larval stage of the adult pine processionary moth.[88] This insect is found throughout southern Europe but especially in the coastal regions of the Mediterranean; it is about 2.5 cm long and is covered with white and reddish-yellow hairs which are poisonous and cause painful inflammation, irritation, and rash.[89] The larvae produce a toxin that contains peptides and perhaps biogenic amines.[90] The caterpillars live in coniferous forests and feed on pines and occasionally cedars; they share a common nest and move around head to tail in columns or processions.[91]

Pliny says that *pityocampae* have the same property as cantharides, that is to cauterize flesh and form scabs, and he adds that they are prepared in a similar fashion (*HN* 29.95). As in the case of the two beetles, the caterpillars are used for skin diseases and as both an emmenagogue and diuretic (*HN* 29.95). The remedies for pityocampa poisoning are sapa (Pliny *HN* 23.62), oil (*HN* 23.80), and milk (*HN* 28.128).

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88 Beavis (1988) 148.

89 Carter and Hargreaves (1986) 201.

90 Green and Siegel (1984) 500.

91 Carter and Hargreaves (1986) 201.

The salamander is the only vertebrate, other than snakes, considered poisonous in antiquity. These lizard-shaped amphibians are covered in thin, scaleless skin which, due to the number of cutaneous glands, is always moist and cold. There are many varieties within the family Salamandridae, but the one referred to in antiquity could be the spotted or fire salamander (*Salamandra salamandra*). This species is patterned with irregular yellow spots and is distributed throughout south and central Europe, Asia Minor, and north Africa.[92] The spotted salamander appears only during the night or after it has rained during the day; otherwise it hides in burrows or under tree stumps.[93]

Pliny describes the salamander as an animal shaped like a lizard and covered with spots; it only appears with great rains, and it disappears in fair weather (*HN* 10.188). The salamander's cool and moist skin seems to have given rise to the myth that the animal is impervious to flames and can even put out a fire. Nicander writes of the salamander's ability to go through fire without pain or injury (*Alex.* 818-21), and Pliny says that the animal is so cold that it puts out fire by its touch (*HN* 10.188). The reproduction of salamanders remained a mystery in antiquity, perhaps because of the amphibian's nocturnal habits and abandonment of its young, which are born in water; Pliny thought that salamanders were infertile and sexless (*HN* 10.189).

The spotted salamander secretes a poisonous substance from its skin glands, especially from the large glands located behind its eyes; this milky, cutaneous secretion, which acts as a deterrent to predators, is

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92 Hvass (1978) 114.

93 Hvass (1978) 115.

highly irritating to human mucous membranes and can be deadly to small animals.[94] The poisonous qualities of the salamander took on grand and dramatic proportions in antiquity, and references to poisoning imply oral rather than topical poisoning. Nicander lists the toxic effects as sudden inflammation of the tongue, chilling, trembling, ataxia and crawling on all fours, blunting of the mind, and livid discolouration of the extremities (*Alex.* 540-5). Scribonius Largus describes similar symptoms: a sore tongue, weakened body, and livid spots (*Comp.* 187). Paulus includes inflammation of the tongue, difficulty in speaking, trembling, torpor, and parts of the body turning livid and dropping off (Paul. Aeg. 5.32). Pliny's account of poisoning is the most colourful. The salamander is the most dangerous of all the poisonous animals since it is able to kill whole communities, unlike other venomous creatures which strike individuals and then die of remorse (*HN* 29.74). Any food or drink touched by a salamander becomes poisonous, and the milky saliva from its mouth (perhaps thought to emanate from the mouth rather than from the nearby glands) has a drastic and far-reaching action; if the saliva touches any part of the body, all the hair on the entire body will fall out, and the part actually touched changes colour and becomes affected by an eruption or psoriasis (*HN* 29.74-5; 10.188).

The depilatory reputation of salamanders may have been due to their corrosive and caustic properties, which Celsus records (*Med.* 5.7.1; 5.8.1). Some of the remedies for salamander poisoning were pine sap mixed with honey (Nic. *Alex.* 546-7; Larg. *Comp.* 187), must (Pliny *HN* 23.29), sapa (*HN* 23.62), oil of oenanthe (*HN* 23.80), and the mixed flesh

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[94] Hvass (1978) 115.

of the sea turtle and the frog (*HN* 32.35). Oddly enough, the cantharid beetle, taken in liquid form, was also considered a remedy for salamander poisoning (Pliny *HN* 29.76).

A toxic substance difficult to categorize is poisonous honey. It can be thought of as an animal product, a plant product, or a food poison. Although it is generally not seriously toxic, naturally poisonous honey can cause alarming symptoms. Poisonous honey is made most often from the nectar collected from the plants belonging to the Ericaceae or heath family, which contains the familiar *Rhododendron*, *Azalea*, and *Kalmia* (laurel) genera.[95] The poisonous principle in the rhododendrons and azaleas is a group of grayanotoxins, while the resin andromedotoxin and the glucoside (a glycoside whose sugar component is glucose) arbutin are the toxic entities in the laurels.[96] The symptoms of poisoning can be varied, depending on the type and quantity of poisonous honey ingested. In general the symptoms are a tingling feeling in the skin advancing to tingling and numbness in the extremities, dizziness, blurred vision, cold sweat, weak pulse, possible loss of consciousness; nausea and vomiting may or may not occur.[97] Patients generally recover between 6 to 8 hours and two to three days after ingestion. The lethal dose of poisonous honey is high,[98] and, due to the bitterness and astringency of such honey and the quantity required, it is unlikely that a lethal amount would be eaten inadvertently. Long term use of rhododendron honey, however, may

95 Patwardhan (1973) 495.

96 Turner (1991) 172 & 82.

97 Patwardhan (1973) 496.

98 Patwardhan (1973) 498.

result in chronic poisoning, which is manifested by constant low blood pressure punctuated by periods of high blood pressure.[99] Honey can also become poisonous if bees take nectar from the flowers of datura and henbane; the poisonous principles in these cases are scopolamine and atropine.[100]

Poisonous honey was recognized and noted in antiquity. Xenophon reports the experiences of soldiers after eating honey in Asia Minor; they all became careless, were afflicted with vomiting and diarrhoea, and were not able to stand up straight (*An.* 4.8.20). Those who had eaten a smaller quantity appeared drunk, while those who had eaten more seemed either mad or about to die; after 24 hours they began to come round and in three or four days they recovered (Xen *An.* 4.8.21). Strabo tells of a group of people called the Heptacometae who weakened soldiers with poisonous honey and then attacked and overcame them (12.3.18). Dioscorides writes of the honey produced in Heraclea of Pontus which can cause sweating due to the properties of certain flowers (*MatMed* 2.103). He also speaks of the bitterness of Sardinian honey (*MatMed* 2.102); this quality is due to the presence of the glucoside arbutin.[101]

Pliny notes that the honey from Heraclea in Pontus is poisonous in certain years; he writes that the poison comes from the flowers of a plant that is toxic to cattle and goats (*HN* 21.74). The plant has been tentatively identified as the common azalea or *Rhododendron ponticum*;

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99 Turner (1991) 172.

100 Patwardhan (1973) 501.

101 Patwardhan (1973) 502.

azaleas, rhododendrons, and laurels are in fact poisonous to livestock.[102] Another kind of honey from the same area of Pontus is said to produce a kind of madness and is made from the flowers of rhododendrons (Pliny *HN* 21.77). Pliny lists the identifying characteristics of poisonous honey: it is thin, reddish, smells strange, causes sneezing, and is heavier than edible honey (*HN* 21.75). Paulus of Aegina made the most astute observations of the effects of Heraclean honey. He says that people who consume this honey have the same symptoms as those who have taken aconite (5.57). Indeed, the symptoms of tingling and numbness in the skin and extremities are similar to those of aconite poisoning, and andromedotoxin affects respiration in a way similar to aconitine.[103] The suggested remedies for honey poisoning in antiquity were all similar. Paulus proposes honeyed wine with rue but also feels that the same remedies for aconite apply, and Dioscorides and Pliny include salted fish taken with the wine and rue (Paul. Aeg. 5.57; *MatMed* 2.103; *HN* 21.76). Pliny also recommends honey in which bees have died as an antidote to poisonous honey (*HN* 29.97).

A few substances believed to be toxic in antiquity can only be classified as imaginary or fanciful poisons since there is no modern evidence of their harmful characteristics. These are Stygian water, bull's blood, and the sea hare.

The Styx was both an Arcadian river, whose source was high in the mountains, and one of the nine rivers of the underworld. The water was said to cause the instant death of anyone who drank it (Pliny *HN* 2.231;

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102 Pliny (1956) 487; Turner (1991) 82, 172.

103 Patwardhan (1973) 499.

31.126), but its colour, smell, and taste gave no clue as to its treacherous qualities (Sen. *Nat.* 3.25.1; Pliny *HN* 2.231). Seneca writes that Stygian water acts with such great speed that there is no time to administer a remedy and that the sole indicator of such poisoning is death (*Nat.* 3.25.1). The water was so potent that it could only be contained in an ungulate's hoof; it would eat through all other containers, even those made of stone, iron, or gold (Pliny *HN* 30.149; Curt. 10.10.16; Paus. 8.18.5). The notion of water having the power to corrode stone or metal containers may have come about from observing rivers and streams flowing through holes in rock or disappearing underground. Since water can gradually wear or dissolve passages through rock, the ancients may have perceived this property as transferable to mere containers. The water draining from Nonacris, a mountain in Arcadia near the Styx, was said to fall to a rock through which it passed on its way to the river (Paus. 8.18.4). Water flowing underground may have given the impression of water turning into stone, which may be the case in Pliny's version; he says that at Nonacris the flowing water itself turns to stone (*HN* 31.27). In large areas of Arcadia rivers and streams do seem to disappear underground because of a lack of surface outlets; the natural drainage areas are below the surface of the earth.[104]

Cold water was thought to be dangerous in antiquity. Pliny felt that the water at Nonacris was harmful because of being particularly cold (*HN* 31.27). Plutarch writes that one version of the events surrounding Alexander's death is that he was poisoned by ice-cold water from

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104 *Ox. Class. Dict.* (1970) 94.

Nonacris, but he adds that most people think that the story is false (*Alex.* 77.4-5). Paulus advises against drinking large quantities of cold water especially after bathing, running, or strenuous exercise (5.65).[105] According to him, the symptoms of cold water poisoning are suffocation and pains, and he recommends opening the veins and administering enemas to counteract its effects (5.65).

Other waters were considered poisonous in antiquity. Pliny mentions deadly waters in Thrace, water at Leontini that kills on the third day after ingestion, and a poisonous spring on Soracte identified by the birds unwise enough to drink from it and lying dead nearby (*HN* 31.27-8). Other strange properties were attributed to certain waters. Seneca tells of two rivers in Boeotia, one of which if drunk by black sheep turns them white, and the other of which if drunk by white sheep turns them black (*Nat.* 3.25.3). Rivers in Macedonia, Galatia, and Coppadocia had similar powers to change the colour of animals (*Nat.* 3.25.4). Although the ancient sources specify Arcadia as the location of the poisonous Styx, the Styx of the underworld and its association with death may have coloured the ancients' view of the properties of this water. Perhaps compounding the superstition was the notion that cold water was dangerous. In the final analysis, it is difficult to believe that this water, from a pure mountain stream, could pose any serious threat to either vessels or human life, except in the case of overheated individuals.

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105 Everyone has heard the anecdote of the athlete or hiker who, after exerting himself on a hot day, drank a large quantity of cold water and died. It is possible that a similar event was observed in antiquity.

Pliny writes that fresh bull's blood is poisonous except at Aegira, where the priestess drinks it before prophesying, and Cicero notes that Themistocles, after drinking bull's blood at a sacrifice, fell dead (*HN* 28.147; *Brut.* 43). The toxic qualities ascribed to bull's blood were that it congealed in the stomach and blocked the breathing passages. Nicander lists the effects of drinking fresh bull's blood as collapse, pain, gasping, convulsions, and foaming at the mouth (*Alex.* 312-8). Scribonius Largus writes that some people who have drunk bull's blood can be identified by the traces of blood left between their teeth; they also experience nausea and choking (*Comp.* 196). Pliny writes that bull's blood is noxious because it clots and quickly hardens in the belly (*HN* 11.222). Paulus lists symptoms such as difficulty in breathing, obstruction of the throat, spasms, a red tongue, and stained teeth with clots between them (5.55). The remedies for this type of poisoning were directed at breaking up the coagulated blood and consisted of vinegar, wild or green figs, soda, rennet, or cabbage seed (Paul. Aeg. 5.55; Larg. *Comp.* 196; Nic. *Alex.* 319-34; Pliny *HN* 20.94; 23.128; 28.162; 31.119). Bull's blood is clearly a mythical poison, as we know that some cultures drink blood as part of their regular diet, and that blood itself is simply not poisonous. Blood could become poisonous if allowed to putrefy, but the ancient sources make it clear that fresh blood was the culprit. However, a real poison added to blood could make the blood itself appear to be toxic.

The sea hare (*lepus marinus*) was supposed to be dangerous to touch, poisonous to some in food or drink, and poisonous to others merely to look at; if a pregnant woman should gaze upon a female sea hare, she was immediately afflicted with nausea, vomiting, and miscarriage (Pliny *HN* 9.155; 32.8). But in India the animal was never caught alive, and just dipping a human finger in the sea was fatal to it (Pliny *HN* 32.9). Speculation about the identity of the sea hare suggests that it is a member of the genus *Aplysia*, either *A. vulgaris* or *A. depilans*.[106] The appellation 'sea hare' is still in use today,[107] and may be a result of the two rhinopores (like tentacles or antennae) that grow above the animal's eyes. The members of the genus are gastropod molluscs sometimes called sea slugs, although they are not true sea slugs. They are charming, unaggressive creatures who retract their heads and tentacles when touched,[108] and they are not at all loathsome. Aelian likens them to snails without their shells (2.45).

The symptoms of poisoning described in the ancient texts are both alarming and amusing. Nicander, Scribonius Largus, and Paulus all report similar characteristics and symptoms. The sea hare tastes like rotten fish; the victim's innards are racked with pain; the urine is crimson and difficult to pass; and the victims cannot tolerate any food, especially fish (Nic. *Alex*. 465-82; Larg. *Comp*. 186; Paul. Aeg. 5.34). Scribonius Largus adds that the patient is nauseous and vomits foam, especially when someone represents a fish or speaks its name. The pitiable sufferer cannot sleep without hearing the sound of waves pounding on the shore. Eventually he becomes the colour of lead and wastes away as if from consumption (Larg. *Comp*. 186). Pliny writes that if people are struck by a sea hare, the first symptom of poisoning is that

106 *A. vulgaris*: Jones in Pliny (1956)16, fn. c; *A. depilans*: *Oxf. Lat. Dict.* (1982) 1018.

107 Purchon (1968) 247.

108 Kandel (1979) 269.

they smell of fish; they die in as many days as the sea hare has lived (*HN* 32.9). The attempted remedies included milk (Larg. *Comp*. 186; Nic. *Alex.* 486; Paul. Aeg. 5.34; Pliny *HN* 28.74; 129; 158), mallow (Nic. *Alex*. 487; Paul. Aeg. 5.34; Larg. *Comp*. 186; Pliny *HN* 20.223), the fresh blood of a goose (Paul. Aeg. 5.34; Pliny *HN* 29.104), and cedar pitch (Nic. *Alex*. 488; Larg. *Comp*. 186). Paulus deemed it a sign of recovery when the victim was again able to eat fish (5.34). In spite of its poisonous qualities, the sea hare had some uses; Pliny suggests that the blood and gall of the sea hare acts as a depilatory (*HN* 32.135), that the ash of the animal applied to plucked eyelids will prevent the hair from growing back (*HN* 32.70), and a gouty foot is relieved after being rubbed with a fresh sea hare (*HN* 32.110).

Today the *Aplysiae* are not considered poisonous, although while alive they are unpalatable to fish, and some of the species eject purple ink when disturbed.[109] *A. depilans*, however, has a poorly developed ink gland.[110] All species, when seriously disturbed, secrete a white, thick mucous (opiline) that, when applied experimentally to fish, jelly fish, worms, and other small oceanic inhabitants, has deleterious effects.[111] Otherwise the genus is insignificant from a toxicological viewpoint. It is difficult to imagine how these innocuous animals achieved such a fierce reputation in antiquity. As for *A. vulgaris*, it does not appear in the literature nor in a comprehensive list of *Aplysia* species. It is possible that this designation is now out of date.

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109 Kandel (1979) 272-3.

110 Kandel (1979) 273.

111 Kandel (1979) 273-4.

To conclude then, Romans probably consumed more lead than what is now considered safe and may have suffered from sub-clinical, chronic lead poisoning. It is likely that lead poisoning affected the lower classes the least. Other metallic poisons were hazards of the work place rather than substances used for intentional poisoning. Of the animal poisons, the cantharid beetle is the most toxic and potentially deadly; the buprestis also contains cantharidin and may have been equally poisonous. For the most part, poisonous honey was probably eaten only by accident and not used as a deliberate poison; the amount needed to cause death is great and the taste too bitter to go unnoticed. The fanciful poisons, bull's blood and Stygian water, are not poisonous in themselves, and the sea hare remains a puzzle.

CHAPTER IV: REMEDIES AND PROPHYLAXIS

Although there was an abundance of potential poisons in antiquity, the remedies were even more plentiful. Based on our modern understanding of poisoning, some of the antidotes had some validity, others were poisons themselves, and still others were purely fanciful. Since the number of palliative measures in antiquity far exceeds the number of poisons, only those substances that can be identified and are useful, interesting, or deleterious will be covered. In addition to devising remedies for poisoning, the people of antiquity tried to protect themselves against poisoning by using oral prophylactics, charms, and amulets, none of which appear to have any real protective properties. In antiquity, belief in one's immunity was probably more important than the substance taken or used.

Before examining ancient remedies for poisoning, it is necessary to understand modern theories and treatment and to dispel the romantic, Hollywood conception of antidotes. Although the idea of ingesting or injecting the contents of a life-saving vial that neutralize the effects of a deadly poison in the nick of time has little basis in real life, it does make for exciting movies and novels. First of all, there is no single universal chemical antidote.[1] Specific antidotes do exist for specific poisons, but these are relatively rare, unavailable except in a few large, urban centres, can only be used when the identity of the poison has been determined with absolute certainty, and are capable of causing secondary poisoning and other harmful effects.[2] Most of the specific antidotes that do exist

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[1] Loomis (1974) 142.

[2] *Poison Management Manual* (1984) 5; Loomis (1974) 142-3; Arena (1979) 47.

are effective only against poisons that are the product of the modern industrial age, such as organophosphate pesticides, nitrates and nitrites, and refined and concentrated narcotics.[3]

The theory of antidotal action is based on four kinds of antagonism. Toxicologically, antagonism means that two substances interfere with one another, or one substance affects the action of the other.[4] Functional antagonism occurs when two chemicals produce opposite physiological effects; for example, if an agent causes a serious drop in blood pressure, another may be administered to raise the blood pressure.[5] The poison is not neutralized, nor does it cease to exert its effect, rather, its effect is counterbalanced by the chemical that produces the opposite physiological response.[6] Unfortunately, this type of antagonism is of more theoretical interest than of practical value because of the difficulty in administering the exact amount of drug necessary to accurately induce the opposite chemical effect.[7] As an example of a real situation, respiratory depression caused by barbiturate overdose is more efficiently dealt with by mechanical breathing apparatus than by any available drugs.[8]

Chemical antagonism, or inactivation, occurs when two chemicals react to form a less poisonous complex. For example, ethylene diamine

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[3] *Pois.Mgt. Man.* (1984) 5.

[4] Doull (1980) 17.

[5] Doull (1980) 17.

[6] Loomis (1974) 141.

[7] Loomis (1974) 140.

[8] Loomis (1974) 140-1.

tetra-acetic acid (EDTA) administered in cases of lead poisoning forms a stable and less toxic complex with the lead; the lead is then unable to harm the body and is readily excreted.[9] Chemical antagonism is the most effective of the four forms of antagonism.[10] Receptor antagonism is initiated when the action of the receptor, or carrier, for the poison is blocked. For example, poisoning by muscarine (an alkaloid produced by a certain type of mushroom) is alleviated by atropine; the atropine does not react with or detoxify the muscarine, but it occupies the space on the receptor that the muscarine needs in order to produce its toxic effects.[11]

Dispositional antagonism involves the inhibition of the absorption of a poison by the body,[12] and it is the essence of both modern and ancient antidotal therapy. The goal of dispositional antagonism is to rid the body of toxic matter as quickly as possible and thus reduce the amount of poison absorbed. Since most deleterious chemicals have no specific antidote, modern treatment for poisoning focuses on limiting the absorption of the poison and life support while the body naturally detoxifies itself and excretes the offending substance.[13]

The primary and most direct method of ridding the body of an ingested poison, and thus limiting absorption, is vomiting. Emesis can be induced by giving the victim syrup of ipecac, which is extracted from a

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[9] Doull (1980) 17; Loomis (1974) 137-8.

[10] Loomis (1974) 137.

[11] Doull (1980) 17; Loomis (1974) 142.

[12] Doull (1980) 17.

[13] Loomis (1974) 143; *Pois. Mgt. Man.* (1984) 1-3.

plant belonging to the coffee family and contains the alkaloids emetine and cephaeline.[14] Ipecac acts primarily as a gastric irritant, and vomiting should occur 15 to 30 minutes after administration. Ipecac itself is poisonous, and, if vomiting does not occur, the alkaloids are absorbed and affect the heart, sometimes fatally. After ipecac has been swallowed, one or two glasses of water should be drunk since emesis is more successful when the stomach is full; milk should not be consumed because it will delay vomiting by protecting the stomach from the gastric irritation caused by the alkaloids. Mechanical inducement of vomiting is not recommended since it results in incomplete evacuation of the stomach contents (only one-third of the volume is expelled compared to other methods). Saline solutions used to induce vomiting may result in fatal salt poisoning (hypernatraemia). Emesis should be avoided if a corrosive substance has been swallowed or if the victim is drowsy or convulsing. The timing of emesis is important because once the poison has passed into the upper intestine it is unaffected by emetic agents.

After vomiting, an adsorptive agent, in the form of activated charcoal, is given to the victim to further reduce the chances of any remaining poison being absorbed. Once the poison is adsorbed it is retained throughout the whole of the intestinal tract; activated charcoal can even overtake and adsorb poisons that have entered the intestines. Charcoal is a black, non-toxic powder with neither smell nor taste; it is produced by the incomplete combustion in the absence of air of

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[14] The general principles of the dispositional treatment of poisoning are discussed in Arena (1979) 42-7; *Pois. Mgt. Man.* (1984) 1-5; Turner and Szczawinski (1991) 10-2; and Loomis (1974) 132-5.

vegetable, animal, or mineral substances.[15] Activated charcoal is treated with steam, which creates a network of small pores; it is this network which increases the adsorptive capacity of charcoal. Charcoal made from wood pulp is porous, has a large surface area, contains little ash, and is about 90 percent carbon; as a result it is an excellent adsorbent. Charcoal made from bone is not as effective because of its high mineral and low carbon (11 percent) content, and charcoal tablets or burnt toast are ineffective. Activated charcoal will not adsorb lead, corrosives, or salt, but it is effective against aconite, atropine, cantharides, hemlock, opium, veratrum, and alkaloids in general.

The final step in reducing the absorption of toxic substances is catharsis, the speeding of the offending matter through the gastrointestinal tract, which decreases the time that the poison is in contact with the absorptive intestinal walls. Modern cathartics are magnesium sulphate (Epsom salts), sodium sulphate, sorbitol (a sugar and alcohol complex), and Fleet Enema (a sodium phosphate-biphosphate complex). Mineral or vegetable oil may be given if the poison is known to be insoluble in fat; otherwise it can enhance the absorption of the poison. Cathartics may be administered either orally or rectally. When as much poison as possible is removed from the body, the patient's vital signs are watched closely and treated as necessary. Generally the heartbeat, respiration, blood pressure, and fluid and electrolyte balance are monitored. In all cases water and electrolytes (lost through vomiting and catharsis) must be replaced.

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[15] Detailed information on the types and uses of activated charcoal may be found in Cooney (1980). A more general treatment is in *Butterworths* (1978) 336 and Arena (1979) 34.

In an emergency situation in non-medical surroundings a popular treatment for poisoning by an unknown substance is the so-called universal antidote (which is distinct from the chemical universal antidote mentioned earlier). The universal antidote consists of two parts charcoal (with burnt toast often substituted for activated charcoal), one part milk of magnesia (magnesium hydroxide), and one part strong tea (tannic acid). As has been seen earlier, burnt toast is an ineffective adsorbent. If activated charcoal is used, either the tannic acid or magnesium hydroxide interferes with its adsorptive powers. The universal antidote is an ineffective treatment for poisoning and should not be used; activated charcoal administered by itself is more useful.

In summary, there are few specific antidotes for particular poisons. Even when an antidote is indicated, conservative treatment of poisoning is recommended since the over-enthusiastic use of antidotes often causes more harm than good. The focus of modern treatment for poisoning is ridding the body of the poison by emesis, administration of activated charcoal, catharsis, monitoring vital signs, and treating symptoms as they arise.

In general, the treatment for the ingestion of poison has changed little over the millennia. The remedies for poisoning in antiquity existed in great number and variety, but the essential first step in ancient antidotal therapy was to rid the body of the poison. The term antidote is derived from the Latin *antidotum* which means something that is "given against" — an antidote or remedy. The term antidote could mean a remedy for a bruise, fall, blow, or pain, but it was used chiefly in reference to poisons (Celsus *Med.* 5.23.1a). The expulsion of poisons was brought about by emesis and catharsis, and the effect of the poison was blunted by the administration of substances such as milk, fatty broths, and oil, which could hinder the absorption of offensive chemicals. Rather than stop at this highly effective treatment for poisoning, the people of antiquity often embellished their basic remedy with decoctions and potions. Although some of these mixtures were probably harmless and may have served to increase the victim's fluid intake, others were either potentially toxic or served to enhance the absorption of poison by the body. One can only assume that in some cases the ancients were operating under the 'more is better' theory, and that the supposed 'remedy' was not recognized as harmful.

The first step in poison management in antiquity was emesis. If the poison was known, one could administer the remedy specific to that poison. If the poison was unknown (the more likely case), immediate action was required; to wait for identifying symptoms to appear was to wait too long (Paul. Aeg. 5.28). According to Paulus, the general regime for any type of poisoning consisted of giving the victim warm oil, either by itself or mixed with water, or a variety of herbal decoctions, and forcing him to vomit (5.28). The ancients appear to have been aware of the necessity of filling the stomach before inducing emesis; vomiting may not occur on an empty stomach, even with the use of strong emetics such as syrup of ipecac.[16] Celsus and Pliny also recommend vomiting as a first step (*Med.* 5.27.11; *HN* 25.128). The combination of warm oil and water or the various herbal decoctions may have been sufficiently nauseating in themselves to cause vomiting, but Paulus recommends

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[16] Arena (1974) 43.

forcing the victims to vomit. In addition, Largus writes that vomiting is induced by a vomiting strap (*lorum vomitorium*) (*Comp. 180*). Nicander recommends putting a finger down the victim's throat (*Alex.* 137-8). As has been seen earlier, mechanical means of inducing vomiting result in only partial or incomplete removal of the stomach contents, but any unexpelled oil in the stomach may, to some extent, have hindered the absorption of the remaining poison. Paulus believed that oil could blunt the effect of poison, but he chose an unfortunate analogy to illustrate his point. He writes that if oil is rubbed on the skin and a caustic substance such as cantharides is applied, then the skin is protected from blistering (5.28). This may be true in the topical application of cantharides, but, if the poison has been swallowed, the oil given as a perceived protection would actually enhance its absorption.[17] In addition to expelling toxic substances, vomiting was also useful for identifying the poison swallowed (vomitus is examined today in cases of poisoning to either determine or confirm the identity of the poison). Paulus writes that vomiting reveals specific poisons which can be identified by the smell, the particulate matter, or the colour (5.28).

After vomiting, Paulus recommends a clyster (enema) to remove any poison that has progressed into the intestines, followed by milk and a variety of substances, herbs, and plants, any one of which may be given in any case of poisoning (5.28). Paulus explains that milk has the ability to change whatever is toxic and harmful (5.28); his theory is not quite right, but milk would coat the stomach, and the fat would inhibit the

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[17] Arena (1979) 405.

absorption of some poisons.[18] Milk and other fatty substances, such as lard, oil, and butter, are frequent components of the poison remedies of antiquity. Pliny recommends women's milk for henbane (*HN* 28.74), mare's milk for the sea hare (*HN* 28.159), cow's milk for all poisons (*HN* 28.160), ass's milk for henbane, hemlock, and sea hare (*HN* 28.158), goat's milk for cantharides (*HN* 28.161), sheep's milk for buprestis and aconite (*HN* 29.105), lard for quicksilver (*HN* 28.158), and Largus, Celsus, and Nicander suggest milk in many of their remedies for specific poisons (Larg. *Comp.* 179;186; Celsus *Med.* 5.27.12a-b; Nic. *Alex.* 205-6; 262-3; 423).

There is little evidence for the use of adsorptive materials in antiquity. One substance in particular that would have been a good adsorbent, yet is mentioned infrequently, is Samian earth. Nicander suggests four drachmas of Samian earth as one of his many remedies for cantharides poisoning (*Alex.* 148-9). One investigator estimates that four drachmas is equivalent to 13.64 grams.[19] Samian earth, also called Parthenian earth, was a white and dense kaolin, or clay, also known as hydrated aluminum silicate, and mined on the island of Samos.[20] Kaolin, taken orally, is an effective adsorbent of toxic materials within the digestive tract and is used in modern medical practice for alkaloid poisoning; medicinal kaolin is commonly known as Kaopectate.[21]

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[18] Doull (1980) 6.

[19] Scarborough (1979) 77. Other experts reckon the drachma to fall between 4.2 and 4.4 g; for the sake of convenience 4.36 g is the generally accepted figure (Lang (1964) 4). Four drachmas at 4.36 g each equals a total of 17.44 g; Scarborough may be using some other measuring system.

[20] Scarborough (1979) 77.

[21] Scarborough (1979) 77-8.

Unfortunately, Nicander's recommended dose of four drachmas would not have been sufficient to alter the course of cantharides poisoning unless only a tiny quantity of the poison had been consumed[22] (and in that case the danger of severe poisoning would be greatly reduced anyway). Dioscorides writes that Samian earth drunk with water is effective against poisons, but he fails to indicate the dosage (*MatMed* 5.172).

Another potential adsorbent in antiquity was Lemnian earth, a clay from the island of Lemnos, which Paulus includes in his list of remedies to be given after emesis and catharsis (5.28). The earth is composed of silicates and oxides of aluminum, iron, and magnesium[23] and may have had an effect similar to Samian earth. Dioscorides also mentions that Lemnian earth is useful against poisons (*MatMed* 5.113), but neither he nor Paulus give any indication of how much of the earth should be taken.

Ash is a recommended treatment in a few cases. Pliny says that the head of a fresh red mullet reduced to ash is an antidote to all poison (*HN* 32.44). Paulus suggests mixing the ashes of wood with the initial dose of oil which is given before emesis (5.28). Ash is the product of complete combustion in the presence of air, while charcoal is the product of incomplete combustion in the absence of air. Since a high ash content is an undesirable quality in activated charcoal and reduces its ability to adsorb poisons, it is reasonable to conclude that ash itself would have little adsorptive capacity and that these suggested ancient remedies are unlikely to have been effective. In addition, the high mineral content in

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22 Scarborough (1979) 78.

23 Thompson (1931) 51.

the bone ash from the mullet would further reduce any adsorptive qualities it might possess.

Apart from enemas administered to effect catharsis, a variety of plants were prescribed which could have had laxative qualities. The plant mallow is often found in ancient remedies for poisoning. The mallow of antiquity is thought to be *Malva sylvestris*, a tall, broad-leafed perennial, bearing pink flowers striped with purple; it is common all over Europe and grows throughout the Mediterranean on stony and neglected ground.[24] The mallow harbours no dangerous constituents, and all parts of the plant, but particularly the leaves and flowers, contain mucilage which acts as an emollient on the gastrointestinal tract.[25] If the mallow is consumed in sufficiently large quantities, it has a laxative effect.[26] Dioscorides writes that a broth of mallow helps in all poisonings (*MatMed* 2.144), Pliny and Celsus suggest mallow for white lead poisoning (*HN* 20.223; *Med* 5.27.12b), and Largus recommends it against the sea hare (*Comp.* 186). Paulus suggests administering a decoction of mallows before forcing a patient to vomit, if oil is not available, or, alternatively, adding the decoction to the initial dose of oil; he adds that the mallow will blunt the effect of the poison (5.28). This action may be due to the laxative effect of the plant or to its mucilage, which could inhibit the absorption of poison.

Betony, which may be *Stachys officinalis*, also figures in ancient remedies. Although modern analysis has been unable to discover any

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[24] Huxley (1977) 103; Polunin (1987) 124.

[25] Bianchini (1977) 86; Schauenberg (1977) 62.

[26] Schauenberg (1977) 62. The author fails to quantify "large".

therapeutic properties in the plant, it remains of interest to herbalists.[27] Betony is a tall perennial with long, oval leaves and purple flowers; it grows throughout western and southern Europe in open areas.[28] Betony contains 15 percent tannin, which acts as an astringent; in large doses betony causes purging and vomiting.[29] Pliny says that, by their laxative action, betony seeds taken in drink remove poisons from the body (*HN* 25.127). Paulus mentions that betony may be taken as a preventative to poisoning, but if poisoning has already occurred, then four to five times as much must be taken, and twice a day rather than once (5.28). Dioscorides writes that betony drunk with honeyed water causes vomiting and that a drachma taken with wine is helpful in cases of poisoning; he adds that betony may be drunk as a prophylactic measure if poisoning is anticipated (*MatMed* 4.1). It is possible that if betony were to be taken in a large enough quantity it could help rid the body of poison by emesis and catharsis.

Linseed, or flax-seed, (*Linum usitatissimum*) is another component of ancient remedies, and, given after vomiting, it would act both as a laxative and as a barrier to absorption. Linseeds contain 30-40 percent oil and about 6 percent mucilage, and linseed oil is employed as a laxative in veterinary medicine.[30] Flax grows throughout Europe on both waste ground and cultivated areas, and the seeds themselves have

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[27] Bianchini (1977) 158.

[28] Schauenberg (1977) 161.

[29] Duke (1985) 457; Schauenberg (1977) 162. Schauenberg does not quantify "large doses".

[30] *Merck Index* (1989) 867-8.

laxative properties.[31] Both Nicander and Paulus suggest that linseed be given before vomiting; Nicander says that it should be administered in a meat broth for cantharides poisoning (*Alex.* 134-8), and Paulus says that a decoction of linseed may be substituted for oil if oil is unavailable (5.28). Largus also recommends linseed for cantharides poisoning (*Comp.* 189), and Dioscorides writes that linseed is a useful laxative (*MatMed* 2.125). One of the constituents of linseed oil is the triglyceride of linoleic acid, an essential fatty acid; if linoleic acid is consumed in large enough quantities it causes nausea and vomiting.[32] It is unlikely, however, that one could consume enough linoleic acid by eating or drinking a decoction of the seeds of flax to cause this effect. In antiquity, linseeds probably acted as a laxative unless vomiting was induced. Furthermore, linseed, or any oily or fatty substance, is contraindicated for cantharides poisoning because oil increases the absorption of this fat soluble poison.

Innumerable concoctions were recommended for use after the body had expelled as much of the poison as possible. Some of these certainly could have acted as laxatives, as has been seen above, others contained tannin, many were probably beneficial only in replacing lost fluids, and some were potential poisons. The decoctions containing tannin-bearing plants are interesting because of the action of tannin. Tannin is an astringent constituent of many plants and is capable of precipitating poisonous materials such as alkaloids, glucosides, and some metals (lead salts).[33] Tannin has been used in veterinary medicine in cases of metal

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[31] Schauenberg (1977) 61; Lewis (1977) 283.

[32] Scarborough (1979) 75-6; *Merck Index* (1989) 867.

[33] Arena (1979) 42.

poisoning.[34] Unfortunately, the tannates formed during the process of precipitation are able to redissolve and release the poison; therefore, they must be removed from the stomach.[35] In addition, the tannin itself can be harmful; it has been demonstrated in experiments that absorbed tannin is toxic to the livers of some animals.[36] Several ancient remedies contain tannin. Paulus mentions betony and nard in his list of suggested medicines to be taken after trying to rid the body of as much of the poison as possible (5.28). Betony, as has been noted previously, contains 15 percent tannin.[37] Nard, possibly *Valeriana officinalis*, contains tannin to a far lesser extent; its more important constituents are alkaloids, valepotriates, and acids.[38] If the nard of antiquity is the same as modern valerian, the remedy would have had a sedating effect on the patient due to the presence of the valepotriates.[39] Only the remedy containing betony would have had a significant enough amount of tannin to have had a binding effect on some poisons, but it should have been administered before emesis in order to ensure the removal of the resulting tannates. The nard probably does not contain enough tannin to affect any poison remaining in the body, but its sedating action might have calmed and relaxed a frightened patient.

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34 *Merck Index* (1989) 1431.

35 Arena (1979) 42.

36 Loomis (1974) 134.

37 Schauenberg (1977) 162.

38 Duke (1985) 503-4.

39 Duke (1985) 503; *Merck Index* (1989) 1558.

Largus recommends myrtle berries or leaves for ephemeron poisoning (*Comp.* 193). Pliny suggests myrtle oil for cantharides, buprestis, and poisons that cause sores (*HN* 23.87); it appears that this remedy was for external use. The myrtle, *Myrtus communis*, is an aromatic shrub bearing black berries; the leaves and fruit contain tannin, oil, resin, and a bitter principle.[40] Dioscorides lists many ailments that can be alleviated by myrtle, but poisoning is not among them (*MatMed* 1.155).

Dioscorides recommends the juice of the root of cinquefoil for countering poisons (*MatMed* 4.42), and Pliny writes that the juice may be taken after vomiting a poison (*HN* 25.128). Celsus says that a decoction of cinquefoil root held in the mouth is a remedy for toothache and that the medicine must not be swallowed, but he does not mention its use as a poisoning remedy (*Med* 6.9.2). Cinquefoil seems to be *Potentilla reptans* (creeping cinquefoil), the rhizome of which contains a large amount of tannin.[41] The rhizome is used in herbal preparations as an astringent and anti-diarrhoeic agent.[42]

Paulus prescribes a decoction of nettle seed to be taken before vomiting (5.28), and Nicander suggests nettle seeds combined with pepper for hemlock poisoning (*Alex.* 201-2). Pliny says that nettle mixed with turtle broth is a remedy for henbane poisoning (*HN* 22.31). The stinging nettle (*Urtica dioica*), common throughout Europe, contains an irritant,

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40 Huxley (1977) 108-9; Bianchini (1977) 158.

41 Bianchini (1977) 62; Schauenberg (1977) 158.

42 Bianchini (1977) 62.

oil, tannin, acids, and various other substances.[43] Some authorities believe that drinking an infusion of nettle leaves can cause mild toxic symptoms such as a burning feeling in the skin, stomach irritation, swelling, and reduced urine output.[44]

It would not be profitable to list all the tannin-containing plants which appear in ancient remedies for poisoning; suffice it to note that many remedies contained varying amounts of tannin and that they were administered either before or after emesis. Most of the ancient decoctions probably did not contain enough tannin to alter significantly the effects of a poison (except perhaps for betony in large quantities). In addition, they would need to be given before vomiting so that the resulting tannates could be expelled along with the poison. Since most of the mixtures seem to be prescribed for use after vomiting, and since their tannin content was probably too low to be significant, it is unlikely that these remedies were as effective as they were thought to be; their therapeutic effects may have been both to comfort psychologically a poisoning victim with the illusion of a medicinal remedy, and to replace fluids lost through vomiting and purging.

Fluid replacement is important in order to counteract the effects of the loss of water and electrolytes through emesis and catharsis, and it is an essential part of the modern treatment of poisoning victims.[45] Dehydration and the concomitant electrolyte imbalance can be fatal if

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[43] Duke (1985) 502; Schauenberg (1977) 280-1.

[44] Duke (1985) 502. Turner and Szczawinski do not include the nettle among toxic plants.

[45] Arena (1979) 47 & 49.

left untreated. Physiologically, an electrolyte is a compound that dissociates into ions in solution. Electrolytes exist in different concentrations in different parts of the body (for example, in blood plasma and in the fluid both within and surrounding the cells) and affect the movement of substances between these sites.[46] The proper concentration and balance of electrolytes is necessary for the normal metabolism and functioning of the body. The essential electrolytes are sodium, potassium, and chloride, but calcium and magnesium are also important. Sodium is necessary for fluid balance, and potassium is involved in the contraction of skeletal muscle but the relaxation of cardiac muscle. Excessive loss of water and electrolytes can lead to a severely reduced blood volume and shock, but the administration of fluids, especially those containing electrolytes, will easily avert this condition.

Most ancient remedies were probably more beneficial as sources of fluids and electrolytes than as direct antidotes to poisons. Milk, in addition to inhibiting the absorption of poison by the gastrointestinal tract, is a source of calcium, which is essential in order for skeletal muscle to relax and cardiac muscle to contract. Paulus (5.28), Pliny (*HN* 28.74;158-9;160), Celsus (*Med* 5.27.12a-b), and Largus (*Comp.* 179;186) all prescribe milk for various kinds of poisoning.

The meat-based broths prescribed as remedies in antiquity are excellent restoratives of water and electrolytes. Pliny recommends goat broth (*HN* 28.160), tripe broth (28.161), chicken broth (29.103), and ram's broth (29.105), and Largus suggests chicken, beef, and sheep broth

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[46] Electrolytes and their function are discussed in *Mosby's* (1990) 411.

(*Comp.* 188;189). Any salt added to the broth (Pliny suggests adding salt to the chicken broth) would increase its value as a source of sodium and chloride. Salt itself, mixed with vinegar and honeyed wine, was another of Pliny's remedies (*HN* 31.104). A mixed broth, such as turtle and nettle (HN 22.31), would also be beneficial since many plants are sources of electrolytes; nettle contains calcium, magnesium, potassium, sodium, and chloride.[47]

Other fluids given in cases of poisoning were the grape-based drinks. Must (grape juice), provided it was unfermented rather than partially fermented, would have helped restore the fluid and electrolyte balance. Pliny recommends must as a remedy (*HN* 23.29-30), but he also suggests sapa (boiled-down must) (*HN* 23.62); as has been seen previously, sapa could contain an unhealthy dose of lead. Wine was a popular remedy for poisoning, but its diuretic effect could exacerbate an electrolyte imbalance.[48] Wine was probably used as a remedy both because of its availability and because of alcohol's long history as a medicinal restorative. Wine was usually prescribed in its pure or undiluted form. Celsus writes that, after vomiting, pure wine should be administered if no other antidote is at hand (*Med* 5.27.11), and both Largus (*Comp.* 179) and Pliny (*HN* 23.43) also recommend undiluted wine. In short, any of the drinks or infusions suggested in antiquity would have been beneficial in helping to restore fluids and electrolytes,

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[47] Duke (1985) 502.

[48] In modern medicine the use of diuretics for hypertension can lead to potassium depletion (*Mosby's* (1990) 411-2).

with the exception of fatty liquids in the case of cantharides poisoning, and diuretics such as wine.

Decoctions made from potentially poisonous plants could, obviously, add to a victim's distress. A popular remedy in antiquity was rue, probably *Ruta graveolens*, a low, aromatic shrub. Nicander suggests rue in wine as one remedy for cantharides poisoning (*Alex.* 154-5), Pliny says that rue is a powerful remedy and an important herb in the making of antidotes (*HN* 20.132), and Largus (*Comp.* 188) and Celsus (*Med.* 5.27.12b) recommend rue for aconite and hemlock respectively. Paulus (5.28) and Dioscorides (*MatMed* 3.52) both suggest the seeds of rue for poisoning in general, and Dioscorides adds that the dose is an *acetabulum* (about one-quarter of a cup) taken in wine. Pliny considers rue to be foremost among medicinal herbs, and he lists innumerable conditions that it can alleviate (*HN* 20.131-43); he also includes its use as an emmenagogue and abortifacient (*HN* 20.130). Dioscorides believes rue to have many uses, including that of emmenagogue (*MatMed* 3.52). Rue contains several significant substances such as alkaloids, furocoumarins, rutin, and oil.[49] The alkaloid philocarpine, found in rue, has been used in veterinary medicine to induce abortion in horses,[50] and rue is used in many parts of the world as an herbal emmenagogue and abortifacient.[51] Rutin is able to strengthen the capillaries and reduce high blood pressure.[52] If rue is taken in large quantities it acts as an

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[49] Duke (1985) 417.

[50] Riddle (1991) 14.

[51] Duke (1985) 417; Riddle (1991) 15.

[52] Duke (1985) 417.

acronarcotic poison (having astringent in addition to analgesic and sleep-inducing qualities); Pliny writes that the juice of rue is a poison if taken in too large a dose (*HN* 20.131). Oil of rue is more poisonous than the leaves and can cause gastric pain, vomiting, convulsions, and even death.[53] It is unlikely, however, that the ancients consumed rue in such a concentrated form, but mild toxic effects could be produced by decoctions of the leaves and seeds.

Another remedy for cantharides poisoning, suggested by Nicander (*Alex.* 144-6), has been identified as heliotrope or *Heliotropium europaeum.*[54] Heliotrope is mentioned by Pliny (*HN* 22.57) and Dioscorides (*MatMed* 4.193) but not discussed as a remedy for ingested poison; it is described as a purgative and emmenagogue. Luckily, heliotrope was not a common remedy, as it is now regarded as unsafe and poisonous; it contains an alkaloid that damages the liver, and it has caused death in livestock.[55]

Both Pliny (*HN* 25.128) and Paulus (5.28) suggest aristolochia as a remedy for non-specific poisoning. Dioscorides describes three kinds of aristolochia and says they are all effective against poison; the dose is a drachma in wine (*MatMed* 3.4-6). The aristolochia of antiquity probably belongs to the genus *Aristolochia* which represents several species of plants called birthwort.[56] Aristolochia contains aristolochic acid which

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[53] Duke (1985) 417-8.

[54] Scarborough (1979) 76.

[55] Duke (1985) 226; Lewis (1977) 56.

[56] Huxley (1977) 73.

is toxic and which has a physiological action similar to colchicine.[57] Birthwort has been used in cases of criminal poisoning.[58]

The last remedy with poisonous qualities worth mentioning is pennyroyal which, fortunately, does not figure prominently in ancient remedies for poisons. Nicander suggests pennyroyal in water for cantharides poisoning (*Alex.* 128-9). Dioscorides mentions no antidotal properties, other than that the herb in wine is good for snakebite, but he adds that pennyroyal is an emmenagogue and abortifacient (*MatMed* 3.36). Pliny's treatment of pennyroyal is similar to Dioscorides' (*HN* 20.152-5). Pennyroyal (*Mentha pulegium*) is poisonous if taken in large quantities due to the presence of a ketone called pulegone.[59] The oil extracted from pennyroyal contains the highest concentration of pulegone; 2.5 ml of the oil has caused convulsions and coma, and 30 ml can be fatal.[60] The oil is an abortifacient, but the dose required to induce abortion also causes irreversible liver and kidney damage.[61] It is unlikely that an infusion of pennyroyal tea would have any beneficial effect on a victim of cantharides poisoning, but if the tea were strong enough, or a large quantity were consumed, the problems facing a poisoning victim could be compounded. On the other hand, an infusion

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[57] Schauenberg (1977) 261.

[58] Lewis (1977) 28.

[59] Duke (1985) 308.

[60] Duke (1985) 308; Scarborough (1979) 75. Modern fatalities from the ingestion of pennyroyal oil are usually the result of young women trying to induce abortion.

[61] Duke (1985) 308.

weak enough to be innocuous could be a valuable source of the electrolytes calcium, sodium, and potassium.[62]

A few remaining remedies can only be labeled as curiosities, and, not surprisingly, they come from the works of Pliny. He writes that the skink (probably a large crocodile-like lizard rather than the modern skink)[63] is excellent in remedies for poisons (*HN* 8.91). If a frog's liver is thrown into the path of ants, that part of the liver that the ants attack is an antidote for all poisons (*HN* 32.50). Pliny also includes the white dung of a hen boiled in hyssop (an aromatic herb) or honey wine (*HN* 29.103), the dung of wild doves (*HN* 29.105), beaver urine (*HN* 32.31), and, seeming somewhat more palatable, diamonds, which have the power to overcome poisons and make them harmless (*HN* 37.61). These remedies serve as illustrations of Pliny's indiscriminate recording of information and the general lack of understanding of poisons and drugs in antiquity.

The notion of protecting oneself from poison by taking prophylactic antidotes began in antiquity and continued beyond the middle of the nineteenth century. The most famous prophylactic was devised by the king of Pontus, Mithridates VI (120-63 BC). Mithridates occupied a great deal of Asia Minor, Greece, and most of the Aegean islands, but the extent of his holdings waxed and waned with the varying degrees of success of Roman action against him. Finally, unable to take a stand against Pompey, and suffering from a revolt against him led by his son, Mithridates tried to end his life with poison, but, because of years of

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[62] Duke (1985) 308.

[63] *OLD* (1982) 1704.

taking prophylactics to preserve himself from poisoning, the poison allegedly had no effect; he was forced to ask an officer to end his life with his sword.[64]

A.E. Housman wrote about the hazards that kings faced and the amazing resistance of Mithridates to poison in a work called *A Shropshire Lad*:

> There was a king reigned in the East:
> There, when kings will sit to feast,
> They get their fill before they think
> With poisoned meat and poisoned drink.
> He gathered all that springs to birth
> From the many-venomed earth;
> First a little, thence to more,
> He sampled all her killing store;
> And easy, smiling, seasoned sound,
> Sate the king when healths went round.
> They put arsenic in his meat
> And stared aghast to watch him eat;
> They poured strychnine in his cup
> And shook to see him drink it up:
> They shook, they stared as white's their shirt:
> Them it was their poison hurt.
> – I tell the tale that I heard told.
> Mithridates, he died old.

The number and identity of ingredients in Mithridates' supposed antidote vary depending on the source, but it is most unlikely that the prophylactic could have conferred any significant immunity to poison. Pliny writes that when Pompey had overcome Mithridates, he found a prescription among the king's belongings — two dried nuts, two figs, and twenty leaves of rue rubbed together with a touch of salt and taken on an empty stomach would protect a person from any poison for one day (*HN* 23.149). Elsewhere, Pliny states that Mithridates' antidote consisted

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[64] *OCD* (1970) 696; App. *Mith.* 12.110-1.

of 54 ingredients, a number that he thinks preposterous, as any one remedy provided by Nature would do (*HN* 29.24). Celsus lists 36 ingredients that make up the antidote that Mithridates took daily to make himself immune to poison (*Med.* 5.23.3a-b). The ingredients consist of a variety of herbs, gums, plant juices, spices, resins, seeds, and roots mixed up with honey; an amount the size of an almond taken in wine is sufficient as protection against poison (*Med.* 5.23.3a-b). The recipe makes about 1.3 kilograms and is enough to last for six months.[65] One ingredient that may be significant is poppy tears, or opium. The quantity called for in the recipe is 17 grams, which, assuming uniform mixing, works out to 94.44 mg of opium per day over 180 days. The alkaloid mainly responsible for the pain-relieving and narcotic effects of opium is morphine, and dried opium contains between 9 and 17 percent morphine.[66] Therefore, 94.44 mg of opium contain 8.5 - 16 mg of morphine, about 1/8 to 1/4 of a grain (one grain is 60 mg). In medicine, 1/4 of a grain of morphine might be considered a normal dose to control pain (although the exact dose is determined by the size and age of the patient), but 1/2 of a grain is a rather large dose. It is necessary to keep in mind, however, that these morphine doses are injected, and in antiquity opium was ingested. The absorption of alkaloids from the gastrointestinal tract can be delayed or inhibited by the gums and other components of opium,[67] and oral doses are simply not as effective as injected doses, so the person taking an oral dose of opium would be

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[65] Watson (1966) 6.

[66] Martindale (1982) 1022.

[67] Beckman (1958) 202; Martindale (1982) 1022.

exposed to less morphine than one who had had an equivalent amount of morphine injected. It is possible that 94.44 mg of opium would have a mild narcotic effect — an effect which would lessen over time unless the dosage were increased. Morphine offers a sense of well-being and freedom from anxiety — both of which could lessen one's fear of poisoning and perhaps create a sense of immunity. Habituation to a set amount of opium might result in some degree of tolerance; therefore, in order to produce a fatality, a higher than normal dose would be required. This tolerance could not confer immunity against any other poison. The other ingredients in the recipe are innocuous and of doubtful use as prophylaxis against poisoning.

A variation on the story of Mithridates is that he drank poison daily, after taking a remedy, to acquire the desired tolerance (Plin. *HN* 25.6). He also used the blood of Pontic ducks, which were said to live on poisons, in his antidotes, and this blood was the most powerful ingredient (Gell. *NA* 17.16.1-3). Mithridates used to show off his immunity by tossing back powerful and fast-acting poisons which appeared to have no effect on him (Gell. *NA* 17.16.4-6). There is little doubt that this story is a folk tale.

In light of its ingredients, Mithridates' antidote offered little protection from poisoning. Although tolerance to chemicals may be acquired through exposure to non-lethal amounts, in many cases a lethal dose has the same effect on both tolerant and normal subjects.[68] The very rapid acquisition of resistance to the pharmacological effects of some chemicals is called tachyphylaxis and is not synonymous with

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68 Goldstein (1974) 569; Loomis (1974) 38.

tolerance. Tachyphylaxis is induced by administering a few doses over a time period of a few hours.[69] For example, if ephedrine (an alkaloid that has a pharmacological action similar to that of adrenalin) is given intravenously to an experimental animal, a rise in blood pressure is recorded. Subsequent doses of ephedrine, given when the blood pressure has returned to normal, elicit less and less of a response until there is no change in blood pressure, even at virtually lethal concentrations.[70]

Tolerance, the decreased responsiveness to a drug, gradually develops by taking habit forming substances such as alcohol, nicotine, and morphine.[71] The observation of tolerance to drugs was made in antiquity. Theophrastus observed that the effectiveness of drugs is reduced in those people who are accustomed to them (*Hist.Pl.* 9.17.1); Pliny echoes Theophrastus and says that the properties of plants confer no benefit to those who have used them daily and that the same theory applies to harmful plants (*HN* 27.144). Sometimes the notion of tolerance is purely anecdotal, as in the case of acquired tolerance to arsenic. More than one hundred years ago certain Austrians used to eat arsenic in gradually higher doses both to produce the illusion of health through a ruddy complexion and to increase their endurance in the mountains.[72] The theory that the gastrointestinal tract absorbs less and less arsenic and thus produces tolerance has not been proven, and there has never been any solid evidence that tolerance to arsenic can be

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69 Loomis (1974) 38.

70 Loomis (1974) 39.

71 Loomis (1974) 39; Glaister (1973) 510.

72 Goldstein (1974) 570.

acquired.[73] Regular doses of arsenic probably only resulted in chronic arsenic poisoning. None of the ingredients in Mithridates' antidote can be considered poisonous (although whether a substance is poisonous or not is dependent on the dose) except for the opium, and that was taken in relatively small quantities. Some degree of tolerance to opium could be acquired through regular use of the drug in significant amounts, but this tolerance would not afford any protection against other poisons.[74] The famous antidote of Mithridates probably had little significant physiological or pharmacological effect.

The most important aspect of Mithridates' antidote was the belief in its effectiveness. Belief must have been the ingredient of primary importance in most of the preventative measures taken in antiquity. The suggested prophylactics have no inherent power to prevent poisoning, and many are simply charms or talismans. Pliny reports that rubbing oneself with certain plants results in immunity; he specifies antirrinum, or wild lychnis, which may be a kind of snapdragon, and euplia, which is an unknown and unidentified plant (*HN* 25.129-30). Wearing either plant on the arm is also effective (*HN* 25.129-30). People who carry artemisia (wormwood or mugwort) with them are not hurt by poisons (*HN* 25.130). Charms placed outside houses could protect the inhabitants. Asphodel planted outside the gates of a villa would afford protection from acts of

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[73] Goldstein (1974) 570; Doull (1980) 415.

[74] Cross-tolerance, the development of resistance to drug A by taking drug B, can be acquired, but opium could not engender tolerance to poisons such as hemlock, aconite, or amatoxin; the drugs, or poisons, must have similar chemical structure and physiological action. Loomis (1974) 39.

poisoning (*HN* 21.108), and a wolf's snout hung on the gates would protect against sorcery in general (*HN* 28.157).[75]

Belief extended beyond charms and amulets to tests for poison. When filled with a poisoned drink, a goblet made of electrum (an alloy of silver and gold) would produce both shimmering rainbows on the surface and a hissing noise like fire (Pliny *HN* 33.81). The association between magical properties and electrum lasted through the Middle Ages and beyond, when it was believed that poisoned foods would tarnish plates made from the metal.[76] Lemnian earth had more than antidotal properties; a cup made of the clay could render a poisonous draught harmless.[77]

In general, the treatment for ingested poison in antiquity was very much the same as modern treatment: rid the body of the offending matter and decrease absorption of the poison by the body. Although antidotes do exist today, they are few, problematic to administer, and are generally specific for poisons that are the product of the modern world. In the ancient world, most decoctions given before emesis served to fill the stomach and increase the effectiveness of vomiting. Since most poisons are emetics themselves, the actual herb or plant used in remedies given before vomiting may have been of little significance. Decoctions given after vomiting helped to restore the fluid and electrolyte balance. It is possible that some of the plants and one mineral may have reduced the noxious effects of some poisons by their laxative, demulcent,

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[75] The wolf appears to have wielded considerable power since horses that stepped in a wolf's tracks became lethargic or unconscious (*HN* 28.157).

[76] Thompson (1931) 51.

[77] Thompson (1931) 43.

or adsorptive natures. Prophylactics such as Mithridates' appear to have contained little if anything that could increase a person's resistance to poisons, and talismans and amulets would have had no effect against poisoning other than to afford the wearer peace of mind.

CHAPTER V: PEOPLE AND POISONS

There is no doubt that poisons and deliberate poisoning existed in antiquity, but the extent of poisoning is debatable and will never be known. Obviously, the choice of poisoning over other kinds of murder was based on a desire for secrecy and subsequent uncertainty as to the cause of death (such motives make even contemporary detection difficult). As a result, the literary sources for poisoning cited in this study cannot be taken at face value. Nevertheless, the sources tell us what was felt at the time to be credible or possible. The motivations for administering or taking poison were varied: revenge, suicide, war, greed, or political manoeuvering. Once a reason for poisoning had been conceived, a murderer had to obtain an effective poison — not necessarily a cheap proposition. An even greater difficulty than obtaining poison was administering it — important figures in Roman society practised vigilance and were often surrounded by protective attendants. Theories existed on how to determine if a person had died from poison, but in reality there was no way of determining the absolute truth; in many cases, if not most, death from poison and death from natural causes were indistinguishable. Many alleged cases of poisoning may well have been natural deaths and vice versa. Poison was perceived as a real and serious threat; laws against poisoning were established, and punishment for the act devised. The purpose of this chapter is to explore the alleged motives for poisoning, the possible methods of acquisition and administration of toxic substances, and superstitions and theories surrounding poisons.

A prerequisite to poisoning is motivation, and revenge may have been the driving force behind some cases of poisoning. A woman is said to have been driven to poison fatally her husband and son because they had killed her son from a previous marriage (Gell. *NA* 12.7.2),[1] and Cicero, in 56 B.C., implies that Clodia brought a charge of attempted poisoning against Caelius because she was angry about the end of their love affair (*Cael.* 69-70). There are several other examples of women who allegedly kill their husbands, although their motives are not always clear. Juvenal suggests that wives are known to poison their husbands (1.69-72), and Apuleius tells of a wife who pays to have her husband poisoned and manages to dispatch the provider of the poison, a doctor, at the same time (*Met.* 10.25-6); he mentions another woman who poisons her husband for the double benefit of adultery and inheritance (*Met.* 2.27).[2] Livy writes of 170 women found guilty of poisoning in 331 B.C.; he does not specify their motives but records that they may have acted out of madness (8.18.10-11).[3] Although it is unlikely that all these

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[1] Much of Gellius' information was second-hand. *OCD* (1970) 460. This particular case took place in Smyrna on the west coast of Asia Minor in the first quarter of the first century B.C. Gellius lived between 130 and 180 A.D.

[2] Even though Juvenal tends to exaggerate for effect, his depiction of the city of Rome, both the general way of life and the physical aspect, are considered historically accurate (Courtney 1980: 19). He uses examples of crime and vice from earlier years to illustrate the activities which were still going on in his time (Rudd 1977: 5; Courtney 1980: 32), so there is an element of truth in Juvenal's writings. Indeed, "... it would be wrong to underestimate the firm foundation of fact which often underlies even the most hyperbolic passages." (Rudd 1977: 6). Apuleius, a novelist, may be working from his imagination rather than from actual incidents. On the other hand, it is always possible that Apuleius is repeating events that really happened, or he may have heard of similar cases and adapted them to suit his own purposes.

[3] According to Livy, after leading citizens had died from what appeared to be the same illness, a slave woman revealed that matrons were preparing poisons. The women were then caught in the act of brewing drugs. Twenty of them, to prove the healing nature of their concoctions, drank the medicine and died. Their associates then informed against the 170 women who were accused of poisoning (8.18.2-11).

examples are true records of poisoning, it is clear at least that Romans believed that domestic, non-political poisoning was a reality.

Although the administration of hemlock was an accepted form of execution in Greece, poisoning does not appear to have been a common method of capital punishment in Rome. A provision in the legal code states that proconsuls, who counted executions among their duties, were not permitted to kill by hacking with an axe, clubbing, impaling, hanging, or poisoning — the use of the sword was preferable (*Dig.* 48.19.8.1). The sentence of death was sometimes ordered by the state to be carried out in the form of suicide, while voluntary suicide was performed to preserve the state or to conform to a code of conduct.[4] Nero ordered Seneca to kill himself (65 A.D.), and although the philosopher first opened his veins, he later drank a draught of hemlock to hasten his death (Tac. *Ann.* 15.61-4).[5] Anteius, condemned for having designs on Nero's empire, also took poison but found it slow to take effect; he slit his veins to ensure his death (Tac. *Ann.* 16.14). During the reign of Tiberius, a man who anticipated condemnation under the law, and who took his own life, did not forfeit his property to the imperial treasury, nor was he denied burial (Tac. *Ann.* 6.29);[6] in this case suicide was considered the equivalent of natural death. This point of law was heavily debated in later years with the result that the wills of those accused or

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[4] Griffin (1986) 192-3.

[5] The hemlock supposedly had no effect. Seneca then got into a warm bath, the vapours of which were said to have killed him (Tac. *Ann.* 15.64). I suspect that the hemlock did eventually work; the poison, combined with Seneca's weakened state from loss of blood, probably caused his death.

[6] It appears that time was of the essence in these cases, and it was necessary to kill oneself before one was formally accused. In the first century A.D., suicide during the trial of a serious case was considered an admission of guilt. Van Hooff (1990) 169.

about to be charged became invalid if the suspected wrongdoers committed suicide (*Dig.* 28.3.6.7; 48.21.3). Suicide also may have been triggered by fear of executioners (Tac. *Ann.* 6.29), and it was a way of avoiding a sentence that may have resulted in a more uncomfortable or humiliating death (Tac. *Ann.* 6.40; 13.30).

There were other reasons for committing suicide in antiquity, and poison was one way of bringing about the desired result. Although suicide was most popular at the end of the first century B.C. and throughout the first century A.D.,[7] it appears to have been a time-honoured method of averting the indignity and enslavement of the war captive. Livy reports that in 211 B.C., 27 senators of Capua committed mass suicide with poison to avoid surrendering to the Romans (26.14.2-5), and that Hannibal killed himself with poison in 183 B.C. rather than submit to the Romans (39.51.7-12; 39.52.8). A group of barbarians, the Cantabrians, who had managed to remain free of Roman rule until 19 B.C., took yew or used the sword to avoid capture (Flor. *Epit.* 2.33.50). Boudicca, in 61 A.D., also chose to end her life with poison (Tac. *Ann.* 14.37). Whether or not these reports are accurate, the historical sources clearly regarded this method of suicide as a likely possibility.

Suicide was an acceptable way of ending a life that had become unbearable. Pliny condones suicide as a release from starvation or weariness of life (*HN* 2.156). In fact, he says that suicide is a heavenly gift (*HN* 2.27), and each man has the power to grant himself a timely

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[7] Griffin (1986) 64. Van Hooff reckons the greatest concentration of suicides took place over a longer period of time during the late Republic and early Empire, from 200 B.C. to 192 A.D. (1990) 234, Appendix B5.

death (*HN* 28.9). Life can become unbearable, and death provides a refuge from ills (*HN* 25.24). Pliny records that the father of Publius Licinius Caecina killed himself with opium to put an end to an intolerable illness (*HN* 20.199). The Digest mentions various motives for suicide; they are a guilty conscience, weariness of life (*Dig.* 3.2.11.3), unbearable pain (*Dig.* 48.21.3.6), and illness (*Dig.* 28.3.6.7). Grief, shame, madness, and despair were other motives.[8] Suicide was even committed for the sake of display, as in the case of some philosophers (*Dig.* 28.3.6.7). The Romans had a rational outlook on the quality of life and saw no reason in prolonging one that had become filled with pain. Indeed, suicide in antiquity was deemed a rational act, in contrast to the modern view of the irrationality of taking one's own life.[9]

The preferred tool for suicide in Roman times was a weapon. According to van Hooff, who has amassed an impressive array of statistics, poison accounted for only 7 percent of Roman suicides, while 56 percent of suicides were performed with a dagger, knife, sword, razor, or scalpel.[10] There must have been reasons for and against the choice of poison for suicide. Seneca (65 A.D.) is suspected of staging an imitation of Socrates' death[11] (although he did try to bleed to death before taking hemlock), while Caecina's father may have opted for the analgesic properties and peaceful oblivion of opium. Martial, however, implies that poison is not quite Roman. In describing the death in A.D. 84/85 of his

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[8] Van Hooff (1990) 83-5.

[9] Griffin (1986) 70.

[10] (1990) 235, appendix B.6.b.

[11] Griffin (1986) 66.

friend Festus, who suffered from an incurable disease, he says that he did not degrade his righteous mouth with poison or subject himself to starvation; he ended his life with a Roman death[12] and freed his spirit by a more noble end (1.78). Even when self-administered, poison retained overtones of stealth and ignobility.

A similar attitude to poison appears to have restricted its use as a Roman weapon of war. A Greek example of the use of poison as a water pollutant in the sixth century B.C. is described by Frontinus. He explains that in one situation the water supply to a town was cut off by the aggressor. Finally, when the people were desperate for water, the supply was turned on, poisoned with hellebore; this poison caused diarrhoea and resulted in a weakened population which was easily overcome (*Strat.* 3.7.6). This story has clear overtones of a theoretical school set-piece, but it is nevertheless interesting that Frontinus or his sources considered the possibilities of using poison on such a large scale. If poison was used in war by the Romans, the resulting victories were hollow. Florus writes that the end of the Asiatic war (around 129 B.C.) was brought about by poisoning the springs supplying several cities. Although the Romans were victorious, they were also shamed, for poison had never before been used officially in Roman warfare (Flor. *Epit.* 1.35.7). The poison is not specified, and it might be difficult to poison a spring, so the accuracy of this account could be questioned. In Tiberius' reign the Romans received an offer (around 15 or 16 A.D.) from the chief of the Chatti to poison the chief of the Cherusci, who were Germanic

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12 This seems to mean that he threw himself on his sword. Griffin (1986) 197. According to van Hooff, Festus had a tumour growing in his neck; van Hooff agrees that a Roman death means death by the sword. (1990) 54.

opponents of Rome. The offer was refused allegedly because Romans fight openly and in arms; the desired result of this refusal was to place Tiberius on the same moral ground as the commanders of old, who both rejected and revealed an offer to poison King Pyrrhus in the third century B.C. (Tac. *Ann.* 2.88). Once again the dark reputation of the act of poisoning is clear, whether or not the incident is reported accurately.

Political advancement was a common motivation for poisoning. The greatest number of political poisonings are reported during the first century A.D., although this may be simply a reflection of the literature available to us. Theoretically, poison could remove potential threats, barriers, and opponents to one's advancement in the political sphere without the use of obvious, overt, incriminating, and politically damaging violence. The cause of death could never be determined with absolute certainty. Tiberius was thought to be behind the death of Germanicus (19 A.D.), using Piso as his instrument (Suet. *Calig.* 2). Tiberius' son, Drusus, was supposedly poisoned (23 A.D.) by his own wife, Livilla, and Sejanus (Suet. *Tib.* 62.1),[13] while one theory concerning the death of Tiberius himself (37 A.D.) is that he was given a slow and wasting poison by Caligula (Suet. *Tib.* 73.1).[14] Nero allegedly knew about the plot to poison Claudius (Suet. *Ner.* 33.1) and was directly involved in the death of Britannicus (55 A.D.);[15] Nero was afraid that Britannicus

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[13] Tacitus reports that Sejanus decided on a slow poison which would mimic a natural illness. The eunuch Lygdus administered the poison (*Ann.* 4.8). It is possible that Drusus died from a disease rather than a poison, especially since his illness was prolonged and had the appearance of disease.

[14] Tacitus does not mention poison in connection with the death of Tiberius. He writes that Tiberius, suffering from ill health, was smothered (*Ann.* 6.50).

[15] Nero wanted to get rid of Britannicus, but since he had no reason to bring a criminal charge against him, and did not dare to kill him openly, Nero ordered that a poison be prepared (Tac. *Ann.* 13.15).

might win greater popular regard than himself on account of his father, Claudius (Suet. *Ner.* 33.2). Since poisoning can wear the mask of disease, the poisoner could hope to dispatch a weak or chronically ill person without arousing undue suspicion. Britannicus was an epileptic, so when he collapsed at dinner after being poisoned, Nero claimed that he was just having another fit (Suet. *Ner.* 33.3). Nero wanted to poison his mother but felt that a second death at his table would look suspicious; in addition, Agrippina seemed to be on guard against treachery, so Nero had to try alternative methods (Tac. *Ann.* 14.3). Poison was also useful for removing potential informers who might divulge damaging information (Livy 39.34.10). Although it is unlikely that all these events involved true poisoning, it is obvious that poison was felt to be an appropriate weapon in these circumstances.

Money and greed have never ceased to be motivating forces for the human race. Cicero conjectures as follows in the trial of Cluentius (66 B.C.): since Cluentius himself had no will, his money, if he died, would go to his mother, the fifth wife of Oppianicus; if she then died, Oppianicus, her husband, would get her property. He alleges that Oppianicus tried to kill Cluentius with poison as a first step to acquiring extra cash (*Clu.* 45). In order to raise money, Caligula (37-41 A.D.) is said to have frightened and intimidated people into naming him an heir in their wills; he, feeling that they were mocking him by continuing to live, then sent them poisoned delicacies (Suet. *Calig.* 38.2). According to Juvenal's gloomy description of Roman life, the desire for money is the root of all crime, including poisoning (14.173-6). The rich could expect to be victims of poisoning by unscrupulous and impatient people waiting for an inheritance. Juvenal accuses one man of poisoning several uncles (1.158), adds that stepsons can expect to be killed (6.628), and cautions wards to guard against mothers dishing out poison (6.629-31), presumably to clear a path to wealth for their own offspring. As one might expect, poverty was believed to be protection against poisoning; Juvenal asserts that the poor had no reason to fear death from poison:

> sed nulla aconita bibuntur
> fictilibus: tunc illa time, cum pocula sumes
> gemmata et lato Setinum ardebit in auro (10.25-27).

Once a motive was felt, a potential poisoner needed to procure a poison. It is unlikely that many individuals possessed the necessary knowledge of poison sources, preparation, and dosages required for a successful murder, and our sources suggest that there were specialists and dealers in poisons and drugs to fill the demand. The occupation was well established before the heyday of poisoning in Rome; Theophrastus (in the late fourth century B.C.) refers to drug vendors and people who make a living harvesting roots (*Hist.Pl.* 9.8.1; 9.8.5; 9.17.1-2). These herb gatherers were a vital source of drugs for the medical practitioners.[16] The growing population and increasing urbanization of Rome itself, and the decreasing amount of arable land close at hand, resulted in the isolation of urbanites from fields and countryside and led to an increased reliance on drug sellers.[17] In addition, the increasing supply of imports, and perhaps a growing demand for new and different drugs, led drug dealers to ignore the herbs and plants that did grow nearby.[18] Rome was well-

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16 Watson (1966) 81.

17 Nutton (1986) 39.

18 Nutton (1986) 39.

poison dealing.

The early years of the reign of Augustus saw increased trade between India and Roman Egypt, and Indian herbs and spices were common in Rome by the first century A.D.[22] Pliny refuses to discuss drugs imported from India, Arabia, or from the outer world (*externi orbis*), since ingredients that grow so far away are unsuitable for remedies (*HN* 22.118). Perhaps he believed such herbs to be insalubrious, or even poisonous. Pliny was convinced of the superiority of Roman herbal remedies,[23] and he claimed that natural remedies were commonly available, without cost, and easy to find (*HN* 24.4). But pre-prepared drugs were probably easier to come by, and Pliny himself admits that most herbs are no longer familiar — that knowledge is restricted to the illiterate country people (*HN* 25.16). Furthermore, why should anyone

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19 Watson (1966) 47.

20 Allbutt (1921) 359 & 350.

21 Beagon (1992) 235. Pliny writes of a man called Demetrius who was prosecuted for trying to drive up the prices in the Seplasia (*HN* 33.164).

22 Scarborough (1982) 140.

23 Beagon (1992) 20.

25 The comments of these three writers, a poet, a satirist, and a novelist of the second half of the first century B.C., the very late first to early second century A.D., and the second century A.D. respectively, although not necessarily indicative of personal experience, do illustrate that poisons and poisoning were a part of the general consciousness or awareness of the Roman world.

hand, Nero is said to have rewarded the efforts of Locusta, after the death of Britannicus, with a pardon for past offenses and a large country estate, and, in addition, to have sent pupils to her to learn her trade (Suet. *Ner.* 33.3).[26]

Even if a poison had been obtained with relative ease, albeit at a price, there was no guarantee that it would work. It might make a victim sick, but not produce the desired fatality. The descriptions of poisons in the earlier chapters indicate that there was not a deep understanding in antiquity of active ingredients, purity of product, and dosages, although some important aspects of poisoning were recognized. Poison was known to be less effective if the victim had a full stomach (Livy 26.14.5), and it was observed that animals and people were affected differently by poison (Lucr. 636-7; 640-1).[27] There is some evidence of poison testing and experimentation on both human and animal subjects, although animals and human beings can react differently to toxic substances. In fact, a problem with modern animal testing of new drugs and foreign substances is the uncertainty of the results if applied to human beings. It is thought that the second century B.C. saw experimentation on animals and people,[28] and Mithridates (120-63 B.C.) is said to have experimented on condemned criminals.[29] The accusers of Caelius (56 B.C.) claimed that

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[26] Not long before Locusta was consulted regarding the murder of Claudius, she had been found guilty on a charge of poisoning (Tac. *Ann.* 12.66). Locusta is said to have come from Gaul and to have been called to court by Nero, whom she instructed in poisoning (Mayor 1966: 119). She was executed by Galba in 68 A.D. (Dio Cass. 64.3).

[27] Livy lived from 59 B.C. - 12 A.D. or 64 B.C. - 17 A.D., and Lucretius probably lived from 94 - 55 B.C.

[28] Allbutt (1921) 347.

[29] Watson (1966) 34.

he tested a poison on a slave, and the swift death which it produced proved the quality of the poison (Cic. *Cael.* 58). Of course, just because Caelius' accusers claim that he poisoned a slave does not mean that he did it. They may have been trying to suggest a familiarity with poisons or merely trying to besmirch his name. Cleopatra is said to have tested an assortment of deadly poisons on condemned prisoners and found that the fast poisons caused too much pain while the less virulent poisons were too slow (Plut. *Ant.* 71.4). As a demonstration of her power to Mark Antony, she killed a prisoner with poisoned flowers dropped in wine (Pliny *HN* 21.12). Tacitus writes that the rapidity of the second poison used to actually kill Britannicus (55 A.D.) was proved beforehand (*Ann.* 13.15), and Suetonius explains that this particular poison was tested on a kid, which lived for five hours, and then, after further cooking, on a pig, which died instantly (*Ner.* 33.3). While it is not possible to verify any of these incidents, the fact of their reportage indicates an awareness of and interest in testing toxicity.

Once a poison had been obtained, there were barriers to its administration. King Perseus of Macedon summarized, in 172 B.C., the major problems surrounding the administration of poison. He said that preparing poison was fraught with difficulty and danger, the preparation required the involvement of too many people, and the result was not guaranteed due to uncertainty about both dosages and the ability to keep the deed hidden (Livy 42.17.6). Tacitus reflects on the unlikelihood of Piso, in 19 A.D., introducing poison into Germanicus' food at dinner, a dinner given by Germanicus, since Piso was surrounded by servants not his own and many bystanders, all potential witnesses; in addition, Germanicus himself, since Piso was reclining next to him, could easily have seen him putting poison in his food (*Ann.* 3.14). If a person had reason to fear for his or her life, then he or she would naturally be on guard against assassination attempts, and the loyalty and incorruptibility of slaves was vital to one's safety from poison in antiquity. A plot allegedly was hatched (after the death of Drusus in 23 A.D.) to kill the three children of Germanicus, but they were protected by faithful caretakers (Tac. *Ann.* 4.12). Nero's mother avoided being poisoned by her son through her own vigilance and the loyalty of her attendants (Tac. *Ann.* 14.3), although Suetonius suggests that she developed immunity against poison by taking antidotes (*Ner.* 34.2). Apparently, subterfuge was not always a requirement in poisoning. In one case that took place near the beginning of Nero's reign (54 A.D.) a Roman knight, Publius Celer, and a freedman, Helius, gave Junius Silanus, proconsul of Asia, poison at a dinner and made no attempt to hide the act; they gave the poison too openly to deceive:

> Ab his proconsuli venenum inter epulas datum est apertius, quam ut fallerent (Tac. *Ann.* 13.1).

Although two professional poisoners are mentioned by Suetonius and Tacitus, it would appear that these women were the suppliers rather than the administrators of poison. Martina, implicated in the death of Germanicus, although this is never stated directly, is referred to by Tacitus as a notorious poisoner (*Ann.* 2.74; 3.7). Locusta is said to have been consulted regarding a poison that would have a delayed fatal effect on Claudius (Tac. *Ann.* 12.66). Locusta supplied the poison, and a eunuch, Halotus, who normally served and tasted Claudius' food, administered the deadly dose (Tac. *Ann.* 12.66). Locusta, according to both Tacitus and Suetonius, provided poison at the request of Nero to kill Britannicus (*Ann.* 13.15; *Ner.* 33.2).[30] Britannicus' attendants had been corrupted, and the poison was administered by his own caretakers (Tac. *Ann.* 13.15). This first attempt was unsuccessful, and Nero either threatened Locusta with execution (Tac. *Ann.* 13.15) or, in another version of the story, he beat her with his own hands (Suet. *Ner.* 33.2). Locusta claimed to have given Britannicus a smaller dose in order to hide the odium of the crime (*facinoris invidiam*) (Suet. *Ner.* 33.2). Locusta promised to supply a swifter and more effective poison (Tac. *Ann.* 13.15). Although a servant always tasted Britannicus' food and drink, the poisoners devised a scheme to overcome this hurdle. A hot drink, already tested by the taster, was given to Britannicus, who refused it as being too hot. Cold water, containing the poison, was added to the drink, and Britannicus' fate was sealed (Tac. *Ann.* 13.16). This incident may be merely gossip and folk tale trickery, but once again it shows a social and political climate in which such acts were believed possible.

Drink seems to have been the most popular medium for the administration of poison. In his defence of Cluentius (66 B.C.), Cicero remarks on the oddity of using bread to conceal poison;

> Iam vero illud quam non probabile, quam inusitatum, iudices, quam novum, in pane datum venenum! (*Clu.* 173).

He goes on to imply that poison given in drink is more easily and thoroughly absorbed by the body and more easily concealed than that given in food. In the case of Claudius, it was suspected that poison had been added to a plate of mushrooms (Suet. *Claud.* 44.2). In this case,

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[30] There is some debate as to whether or not Locusta was actually involved in the deaths of Claudius and Britannicus. Bradley (1978) 198-9.

poison mushrooms could easily have been added to and hidden amongst the edible varieties, but cooked mushrooms themselves are not an ideal vehicle for added poison (it would be difficult to disguise the taste and/or smell of many toxic substances; in addition, the poison might be visible if used in a powdered form). Nero gave poison to his freedmen in both their food and drink (Suet. *Ner.* 35.5), and Vitellius is said to have given poison with his own hand in a glass of cold water (Suet. *Vit.* 14.1).

Whatever the medium, poison obviously had to be concealed and slipped past the tasters, unless it happened to be amatoxin, which has a latent period of about 8 hours. Colchicum also has a significant latent period — symptoms appear after 3-6 hours and can be delayed 12-48 hours. Paulus explains how the taste of a poison can be disguised with sweeteners and the smell with aromatic substances (5.27). Poison may be concealed in medicinal preparations or in prepared food; therefore, it is advisable for someone afraid of being poisoned to avoid such prepared dishes and intense tastes such as sweetness, saltiness, and acidity (5.27). There seems to have been an understanding of such techniques in earlier times. Seneca is said to have avoided being poisoned by Nero by drinking only water and eating simple, unprocessed foods (Tac. *Ann.* 15.45). Nero supposedly sent Burrus poison under the label of throat medicine (Suet. *Ner.* 35.5). Tacitus' version claims that Burrus was already afflicted with a swelling of the throat which blocked the passage of air, so that his death could have been due either to natural causes or to poison smeared on his throat in the guise of a remedy (*Ann.* 14.51). In this account we have a straightforward statement of the difficulty of determining whether or not a death was accidental or the result of poisoning.

According to the lore of antiquity, a tell-tale sign of poisoning was a livid, or black and blue, corpse. The precise meaning of livid must be clarified, since its definition has changed over the millennia. In Latin, the adjective *lividus* indicates a grey-blue or slate colour, the sort of discoloration produced by bruising. The noun *livor* refers to the actual discoloration of the skin. The *Oxford English Dictionary* confirms that the modern meaning of the word "livid" has maintained its ancient meaning, but it also says that the word used colloquially means "pale with rage" — the complete opposite of black and blue. Webster's dictionary states that the primary meaning of livid is discoloured as if by bruising, black and blue, or leaden, but an alternative meaning is ashen or pallid. Modern medical writers rarely refer to lividity and poisoning, but when they do it is difficult to know whether they mean pale or black and blue. I believe that they mean an ashen appearance rather than leaden, while the ancient writers clearly meant black and blue or a slate colour.

Cicero writes that if a body swells and becomes livid, then it is a sign that the person was killed with poison. (*RhetHer.* 2.8). Suetonius, writing about the death of Germanicus, says that poison may have been used since there were bluish discolorations (*livores*) all over his body, and foam flowed from his mouth (*Calig.* 1.2). According to Tacitus, when Germanicus' body was exposed it was uncertain whether or not he showed signs of poisoning (*Ann.* 2.73). One of the ancient characteristics of aconite was that it made the limbs go livid immediately (Larg. *Comp.* 188), as did hyoscyamus (Larg. *Comp.* 181). A livid fingernail was a places where the body is in contact with a hard surface; the weight of the body blocks the blood vessels and prevents them from filling.[37]

Another belief in antiquity was that the heart of a person killed with poison would not burn. Unfortunately, this trait was also attributed to those who died from heart disease (Pliny *HN* 11.187); therefore, even if the flesh were fire resistant, an unsinged heart could not stand as evidence for murder by poison. The origin of the notion of poisoned flesh being impervious to flame can only be guessed at; perhaps the heart of a person killed by foul and devious means maintained its purity and innocence, and proved it by remaining unscathed on the pyre.[38] On the other hand, all the stories claiming that poisoning was easily detected may have been cautionary tales designed to act as deterrents to potential poisoners.

In reality, there was no way in antiquity of accurately determining whether a death was due to poison or disease. An acute illness could appear to be a case of poisoning because of symptoms such as vomiting, diarrhoea, dizziness, weakness, or sudden death; conversely, poisoning could appear to be an acute illness. In addition, there is always the possibility that acute poisoning was caused by bacterial contamination of food, and this could be an explanation for perceived mass poisoning after banquets. The possibility of confusion between death from poison and death from natural causes was acknowledged in antiquity. Livy

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[37] Glaister (1973) 115; Simpson (1980) 132.

[38] By touching King Pyrrhus' right big toe, one could cure oneself of disorders of the spleen. When the king, who lived from 319-272 B.C., was cremated, his big toe could not be burned, so it was put into a box for storage (Pliny *HN* 7.20). Obviously, there was some connection in antiquity between fantastic powers and non-inflammability.

relates that a particular year (331 B.C.), infamous for the disease and pestilence which caused the deaths of many leading citizens, may in fact have been a year of mass poisoning (8.18.2).[39] Cicero was aware of the difficulty in determining the cause of death and used this uncertainty in his defence of Cluentius. To the fact that Oppianicus died suddenly, he replied that this was the lot of too many people to suspect poisoning in this case (*Clu.* 174). Tacitus records the death of a man exiled from Rome and says that it was a point of debate whether he died naturally or from poison, the answer depending on what each person wanted to believe (*Ann.* 12.52). Even in relatively modern times poisoning may be diagnosed as a disease; chronic lead poisoning was thought to be endemic colic until the mid-nineteenth century.[40] Poisoning from the use of lead-glazed crockery continues to be misdiagnosed even now.

Although death from poison and death from natural causes can be easily confused, and the truth cannot be absolutely determined without the scientific tests which were unavailable in antiquity, poisoning certainly took place. The existence of toxic substances was acknowledged, poisons were available for purchase, and historians and other Roman authors assume that these were in fact put to use. Eventually, poisoning engendered specific laws. Livy claims that before 331 B.C. there had never been a trial for poisoning in Rome, but in that year 170 matrons were found guilty of mass poisoning (8.18.10-11). In 174 B.C. a woman who was charged and convicted of killing her husband

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[39] The most likely cause of inexplicably high rates of death in antiquity is infectious disease.

[40] Stevenson (1959) 15.

with poison went into exile (Livy 41.25.6). Sulla is credited with devising the first law dealing with poisons and poisoners. This law was established in 81 B.C. to address assassination, including poisoning.[41] The *lex Cornelia de sicariis et veneficis* deemed it a punishable offence to make, sell, or possess poison for the purpose of murder (*Dig.* 48.8.3). The punishment for a conviction under this law was deportation to an island and confiscation of one's property. A later penalty was capital punishment unless the criminal was lucky enough to be of high status;[42] the lower orders were either crucified or thrown to the beasts (*Dig.* 48.8.3.5).

These particular punishments may have served as guidelines only. When Galba was a provincial governor, he crucified a Roman citizen for poisoning his ward, whose property he stood to inherit. When the man pleaded the law and his citizenship, Galba pretended to alleviate the harshness of the punishment by putting the man on a cross which was whitewashed and higher than the others (Suet. *Gal.* 9.1).[43] Seneca, in his *Controversiae*, states, hypothetically, that a woman who administers poison is to be tortured until she names her accomplices (9.6). This

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[41] Berger (1953) 550. Nero, contemplating the murder of Britannicus, claimed that he was not afraid of the Julian law (Suet. *Ner.* 33.2). Apparently there was no Julian law concerned with poisoning; Suetonius is thought to have made a mistake here (Bradley 1978: 199). Since it is unlikely that Suetonius worked from any sort of index system and had to rely a great deal on memory, any "imprecisions, errors, and omissions" are understandable (Wallace-Hadrill 1983: 15).

[42] The death penalty existed in the laws of the late Republic, but it was probably not used against citizens. During the Empire, citizens were less safe, and anyone convicted of treason, at least, could expect capital punishment, regardless of status (Garnsey 1970: 105).

[43] Crucifixion was usually reserved for slaves. The fact that the victim invoked the law and his citizenship does not imply that a law existed forbidding the crucifixion of citizens, "but rather that the punishment was properly and normally employed against slaves and perhaps humble aliens." (Garnsey 1970: 127).

method of extracting information from a poisoner does not appear in the *lex Cornelia.* During the Republic, free men, in general, were not tortured, but by Augustus' time only Roman citizens were free from the threat of torture.[44]

Although topical poisons were feared in antiquity, there is little evidence either that a substance existed sufficiently potent to penetrate, unnoticed, the barrier provided by the skin, or that this was a common method of applying poison. Aconite can penetrate the skin and cause poisoning, but the amount needed could not be applied surreptitiously to the victim. It is conceivable that aconite could be mixed with oil and applied in a massage, but both the masseuse and the victim could not fail to notice the numbness that aconite causes. Cantharidin can also be absorbed by the skin, but it causes blistering and sores which would alarm and hurt a potential victim. Topical poisoning requires physical contact with a victim and does not offer the extra security of arms-length poisoning through comestibles.

The fear of poisoning through the topical application of toxic substances is reflected in the *lex Aquilia,* which states that anointing or smearing someone with poison is an actionable offence (*Dig.* 9.29.1). The idea of poisoning by skin contact has a long history in the mythology of antiquity. Both Medea and Deianira were famous for their poisoned robes, but in reality there did not exist a poison that was powerful enough to poison through clothing. Examples of modern topical poisons

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[44] Garnsey (1970) 143. Any person charged with treason, however, might be tortured as a punishment or for information; in contemplating or attempting a treasonable act, a person gave up all rights and "could be treated as a slave", i.e., tortured. Garnsey (1970) 145.

are nicotine, which is extremely toxic (a small amount is easily and quickly absorbed), carbon tetrachloride, and some pesticides. Fear of poisoning through skin contact still exists, perhaps legitimately; opponents of Dr. Banda of Malawi have been warned that if they send their suits to the dry-cleaners, they may come back impregnated with poison.[45]

It is difficult to summarize general trends in poisoning since so much of the literature of antiquity has been lost. Furthermore, poison and poisoning may have been taboo subjects at certain periods and considered too dangerous to discuss openly. From the information that is available, the first century A.D. stands out as the most popular period for the use of poisons. Suicide and the use of poison to achieve political ends are the two main motivating factors. Poisoning inspired by greed occurs in uncertain numbers from the first century B.C. to the second century A.D. Again, because of the limitations of the literature, specific numbers cannot be determined; many of the references refer to being poisoned for money as one of the hazards of being rich, but they do not cite specific examples. Domestic poisonings are also difficult to quantify, but they tend to be scattered in terms of time and motive. The example of the 170 matrons of Rome found guilty of poisoning in 331 B.C. makes no mention of a possible motive. Poisoning of husbands, sometimes out of revenge or jealousy, continues in a desultory manner to the second century A.D. The use of poison as a chemical weapon in war is rarely mentioned; as has been seen previously, such a devious and cowardly

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[45] "Poison fears," *MGW* (1992) 6. Apparently the CIA dreamed up a similar scheme as a way of ridding the world of Fidel Castro.

means of overcoming an enemy was not considered worthy of the Roman soldier.

Obstructions to political and financial success were often removed by poison in antiquity. Poisoning was also a non-violent method of ending one's own life in cases of present or anticipated hardship. The rich and powerful, or heirs to coveted positions, were liable to be poisoned, and indeed access to poisons was, for the most part, restricted to those who could afford them. It is possible, however, that country-dwellers of limited financial means might have some knowledge of poisonous plants and substances and could acquire them at no cost. Many cases of suspected poisoning were just that; there was always a residual uncertainty as to the cause of non-violent death in antiquity due to the similarity between the symptoms of poisoning and death from natural causes. Laws were established in an attempt to address the problem of poisoning, but their efficacy is difficult to establish. Toxic substances were recognized, and people were certainly poisoned in antiquity, but considering the difficulties and barriers that had to be overcome in order to pull off a successful poisoning, the real extent of the activity remains unknown.

In summary then, the botanical world provided the greatest abundance of drugs and poisons in the ancient world. Aconite appears to have been one of the best choices for murder because of its potency and rapid action. The *Amanita* mushrooms would have been an excellent murder weapon because of their innocuous taste and delayed action; however, unless dried and stored, their limited availability might have restricted their use. The autumn crocus was another useful poison because of its delayed action. An overdose of opium could provide a painless suicide. Although not all toxic plants were suitable for deliberate poisoning, there were probably cases of accidental death from overdoses or complications arising from draughts which were meant to have beneficial effects.

Of other toxic substances in antiquity, lead was the most insidious. The Romans were probably exposed to low levels of lead in food and drink and may have suffered from sub-clinical, chronic lead poisoning. The symptoms of this condition are vague and non-specific, but significant amounts of lead have been found in skeletons in Roman Britain. It is possible that people in other parts of the Roman world were exposed to similar amounts of lead. Of the animal poisons, the cantharid beetle, and possibly *buprestis*, provided the most toxic substance, cantharidin. The best way to administer a lethal dose of cantharidin would have been in a heavily flavoured drink which was meant to be swallowed quickly rather than sipped. The imaginary poisons were just that and belong to the realm of folklore.

The most effective treatment for poisoning was recognised in antiquity. Vomiting, followed by non-diuretic drinks and infusions, would expel some of the poison and replace essential fluids and electrolytes. Drinks with laxative or demulcent properties might reduce the absorption from the gastrointestinal tract. The usefulness of antidotes taken as a precaution against poisoning was limited; their most important function was to offer a person confidence and peace of mind. The same may be said of talismans and amulets.

Reasons for the use of poison in antiquity varied from suicide, voluntary or forced, to greed, to political manoeuvering. Poisons may have been obtained from drug vendors, from professional poisoners, or by personal acquaintance with drug and poison lore. Giving a poison was probably trickier than getting it; bitter tastes or smells had to be disguised, and, in the case of august personages, the poison had to get past tasters and watchful attendants. A toxic substance could be administered in the guise of a medicinal remedy. As is to be expected from a lack of scientific understanding of the principles of toxicology, poisons and poisoning were imbued with superstitions. In addition, the lack of understanding of diseases and the disease process led to confusion between deaths from poison and deaths from acute illness. Many cases of perceived poisoning, especially mass poisoning, were most likely deaths due to disease.

The best ancient sources for poisons are the medical and pseudo-scientific writers, although it is not always easy to clearly identify the plants and poisons in modern terms; one occasionally must rely on guesswork and probability. Information on specific cases of poisoning is found in the works of historians and biographers, but there is no guarantee that they are recording facts; often the material has been altered by its passage through many hands, and little of it is based on first-hand knowledge and observations. Furthermore, several authors can give completely different accounts of the same event. Although they may be anecdotal in nature, the comments of satirists and novelists do indicate that poisoning was a part of the Roman world. Poets, in general, provide little solid material, since they tend to use poison metaphorically. Even though some ancient sources are suspect, it appears that potent and effective poisons were recognised and used, and their existence acknowledged in the ancient world.

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VITA

Surname: Richards Given Names: Elizabeth Jennifer

Place of Birth: New Westminster, B.C. Date of Birth: Nov. 20, 1957

Educational Institutions Attended:

University of Victoria	1975 to 1979
University of Victoria	1984 to 1986
University of Victoria	1989 to 1994

Degrees Awarded:

B.Sc.	University of Victoria	1979
B.A.	University of Victoria	1992

Honours and Awards:

President's Scholarship (for part time studies)	1985
Catriona Cameron Bingham Prize	1991
Edward B. Paul Memorial Scholarship	1991
UVic Faculty Scholarship	1991
President's Scholarship	1991
Graduate Teaching Fellowship	1992-94
Dean's Scholarship	1992-94

Publications:

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Author: [redacted]
(Signature)

ELIZABETH JENNIFER RICHARDS
(Name)

April 28, 1994
(Date)