Essential oils and hypertension - is there a problem?

Robert Tisserand

Summary
Many aromatherapy books and training courses list essential oils that are contraindicated for people with high blood pressure, such as peppermint, eucalyptus, black pepper, pine and many others. A 1964 French book by Jean Valnet mentions hyssop, rosemary, sage and thyme oils as being hypertensive. However, a close examination of the original 1940s research reveals that, if anything, these essential oils reduce blood pressure. In other essential oils, the effects on vascular tone depend on whether they are ingested, rubbed on the skin, or inhaled. Ultimately, there is currently no compelling evidence that any essential oil is dangerous to use in hypertension.

Introduction
We live in a time of sometimes-strident calls for convincing, clinical evidence that alternative medicines such as aromatherapy are effective. This is a reasonable expectation. In just the same way, if an assertion is made that an essential oil is unsafe in a particular context, we should expect to see compelling supportive evidence. We also live in a time of escalating legislative challenges to essential oil use and availability (some of which are in fact not based on compelling evidence), and it does not make sense to add to this burden by allowing restrictions to be put in place that are based more on fear of legislation than factual evidence.

Blood pressure
Many aromatherapy books and training courses list essential oils that are contraindicated for people with high blood pressure, but the basis for this information is elusive. And while there is some degree of consensus, the list of contraindicated oils varies between sources. As far as I know, no comprehensive review of essential oils and blood pressure has ever been published.

Blood pressure (BP) changes normally throughout a daily cycle, and can vary with mood, activity, and body position. However, persistently high BP is a problem, as it can lead to cardiovascular and/or kidney disease. In a hypertensive individual, small fluctuations in BP are generally not harmful but prolonged, moderate increases can be, especially if they occur on a regular basis. Smoking, poor diet, obesity, and lack of exercise can all contribute to such increases. An
essential oil that causes a sustained, moderate increase BP is therefore a potential problem for someone with hypertension.

**Box: Selected websites and contraindicated essential oils for high blood pressure**

<table>
<thead>
<tr>
<th>Essential Oils</th>
<th>URLs</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><a href="http://www.allthingszen.com/aromatherapy.php">http://www.allthingszen.com/aromatherapy.php</a></td>
</tr>
<tr>
<td></td>
<td><a href="http://www.uklupus.co.uk/aroma.html">www.uklupus.co.uk/aroma.html</a></td>
</tr>
<tr>
<td>Rosemary, Sage, Thyme, Pine</td>
<td><a href="http://www.herbalfitness.com">http://www.herbalfitness.com</a></td>
</tr>
<tr>
<td></td>
<td><a href="http://www.aromatherapy-at-home.com/Essential-oils-high-blood-pressure.html">http://www.aromatherapy-at-home.com/Essential-oils-high-blood-pressure.html</a></td>
</tr>
<tr>
<td>Rosemary, Sage, Thyme, Eucalyptus, Cypress, Ginger, Rose</td>
<td><a href="http://www.holisticshop.co.uk/articles/essential-oils-guide">http://www.holisticshop.co.uk/articles/essential-oils-guide</a></td>
</tr>
</tbody>
</table>

The Table shows eight essential oils that are listed as being contraindicated in hypertension in selected aromatherapy texts over a 40-year period. There is a clear tendency to repeat what others have said, apparently without checking the validity of the information. The essential oils listed in the Box were culled from various websites in January 2010, and is necessarily only a snapshot. I have organized it to show a progression - the list getting gradually longer - because I think this reflects what has actually happened. The “hypertension hotlist” gets stranger by the day. If you Google “pine” or “ginger” and “blood pressure” for example, you will find that both these plants are actively used to *reduce* BP. How cypress, ginger and rose made it onto a hotlist I have no idea. I was also puzzled to see pine oil, until I saw this:
“There is a long standing, practically universal contraindication for some oils in cases of high blood pressure. Hyssop, Rosemary, Sage, Thyme and Eucalyptus are commonly sighted [sic] but just about any stimulating oil such as pine could also qualify. In the absence of critical studies with scientific overview it is wise to err on the side of caution and avoid stimulating oils in cases of chronic high blood pressure.” [http://www.auracacia.com/asktheexperts/?p=89](http://www.auracacia.com/asktheexperts/?p=89)

If stimulating oils can raise BP, then we need to know which ones these are. Very few essential oils or constituents are central nervous system (CNS) stimulants, though some increase alertness through the autonomic nervous system (ANS). Inhaled peppermint oil, for example, increases alertness, but it should not be assumed that it can therefore raise BP, especially since topically applied menthol reduces BP. So if there is a connection with stimulation, it’s not a simple one.

Let’s go back to 1964 and see if we can figure out how we got into this mess.

**The four “Valnet oils”**

The essential oils most commonly cited in aromatherapy texts as being contraindicated in hypertension are hyssop, rosemary, sage and thyme (see Table). It seems all but certain that the original source of this information was Valnet (1990), first published in French in 1964. In this text, these four essential oils are the ones said to be hypertensive, and in each case Valnet gives the same two references. One is Caujolle and Franck (1944), but this is a mistake, since the paper concerns lavender, lavandin and spike lavender oils only. (He probably intended to cite one of the Caujolle and Franck 1945 papers.) The other reference is a thesis by R. Cazal, published in 1944, and I was not able to locate a copy of this. So, one incorrect reference, and one obscure one. But below is some information I was able to find.

**Table: Essential oils reputed to raise blood pressure in aromatherapy texts**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Essential oil</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black pepper</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>✓</td>
<td>-</td>
<td>✓</td>
</tr>
<tr>
<td>Camphor</td>
<td>-</td>
<td>✓</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Eucalyptus</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>✓</td>
<td>-</td>
</tr>
</tbody>
</table>
When 1 g/kg body weight of alcohol saturated with sage oil was given iv to dogs there was no increase in BP, and in some cases a slight fall occurred (Caujolle and Franck 1945a). Similarly, an aqueous-alcoholic extract of sage caused a moderate but prolonged hypotensive effect in cats when given iv (Todorov et al 1984). A hypotensive action has been reported for thyme oil in the abstract of a Russian paper (Kulieva 1980). This is consistent with the calcium channel blocking (and therefore hypotensive) actions of its major constituents thymol and carvacrol (Magyar et al 2004). Intravenous carvacrol is hypotensive in rats (Aydin et al 2007), and topically applied thymol is hypotensive in rabbits (Futami 1984). So, all the evidence suggests that sage oil and thyme oil reduce BP.

I could find no information on rosemary oil and BP, but 1,8-cineole, a major constituent of most rosemary oils, is hypotensive. It caused a 25% fall in systolic pressure when administered iv to dogs in an emulsion at 26.3 mg/kg (Northover and Verghese 1962). Similarly, intravenous 1,8-cineole was dose-dependently hypotensive in rats at 0.3-10 mg/kg (Lahlou et al 2002). Many rosemary oils also contain substantial amounts of camphor. In cats, camphor caused an initial fall in BP lasting 2-10 minutes, followed by a rise of 8-30 mm Hg above normal and lasting for up to 30 minutes, when given intravenously at only 5 mg/kg (Christensen and Lynch 1937). Whether camphor is hypotensive or hypertensive has been debated for more than a century. It is worth noting that spike lavender oil injected iv in dogs caused a slight and short-lived reduction in BP (Caujolle and Franck 1944). Worth noting because spike lavender oil typically contains 31% 1,8-cineole, 17% camphor, and in that sense is similar to many rosemary oils. Even the camphor chemotype of rosemary has approximately the same amount of 1,8-cineole as it does camphor.

But why would Valnet list these oils if there is no evidence that they can raise BP? Well, there is at least some information on hyssop oil, and the explanation of the effect is intriguing. When injected iv in convulsant doses, both
Hyssop oil and wormwood oil produced a sudden drop, then rise in BP, the convulsions coinciding with BP elevation. After extensive testing in cats, Coombs and Pike (1931) concluded that wormwood oil produced a hypotension that initiated the seizures, and that the muscular contraction of the seizures caused the spike in hypertension. BP dropped below baseline after a seizure, and did not rise until the next seizure took place. Similarly, injecting dogs iv with 1-2 mL of a solution of hyssop oil in alcohol resulted in an initial fall in BP, followed by a rise, which was accompanied by seizures. Both the seizures and the rise in BP lasted 3-4 minutes (Caujolle & Franck 1945b).

Whether the seizures were caused by an initial hypotension is debatable, since we now know that the convulsant action of wormwood and hyssop oils is linked to the GABA antagonistic action of their respective constituents thujone and pinocamphone. However, hypotension can cause seizures, and the effects seen on BP may be in part a function of the sudden influx of essential oil into the blood. Hyssop oil and wormwood oil are CNS stimulants, but in spite of this they are hypotensive. (Valnet would not have mentioned wormwood oil, simply because it was not profiled in his book.)

It should be noted that sage oil can cause convulsions, and that it contains both thujone and camphor, yet a fairly massive dose of sage oil was hypotensive (Caujolle and Franck 1945a). (Sage oils contain varying amounts of camphor and 1,8-cineole, and in some oils, the 1,8-cineole content is higher than the camphor content.) In the same report, iv administration 1-2 g/kg of clary sage oil in dogs caused a slight elevation of BP (2-4 cm Hg) that lasted 45-60 minutes. This is curious, since clary sage oil contains 60% linalyl acetate, 15% linalool, and both of these are CNS depressants. Also, lavender oil (40% linalyl acetate, 37% linalool) caused a reduction of BP in dogs when administered in the same way and in the same dose (Clerc et al 1934). So, why clary sage oil would be hypertensive is puzzling.

Could Valnet have confused sage oil and clary sage oil? We know he made a mistake with one of his references. Even if the missing Cazal thesis shows all four oils causing hypertension in dogs, I suspect this has little relevance to aromatherapy practice. It is also worth noting that some of the early reports, especially the French ones, were very loosely written, lacking the kind of detail seen in papers today.

The other oils
The other oils listed in Table 1 are black pepper, camphor, eucalyptus and peppermint. I could find no information on peppermint oil and BP, but topically applied menthol reduces BP for a short time through an effect on local tissues (Futami 1984, Ragan et al 2004), and the calcium channel blocking effect of both peppermint oil and menthol makes a hypertensive action unlikely (Hills and Aaronson 1991). Similarly, eucalyptus oil is 75% 1,8-cineole, which we know
is hypotensive. As for camphor oil, I listed it because at the time I thought camphor (the constituent) was hypertensive, and that white camphor oil was high in camphor. In fact it contains approximately 2.5% camphor, and 40% (+)-limonene. Black pepper oil contains about 20% (+)-limonene, which lowered BP when injected iv into rats (Touvay et al 1995). On this basis, all these oils are more likely to be hypotensive than hypertensive.

**Inhalation**

Temporary hypertension has been recorded following the inhalation of certain of essential oils. A Slight increase of systolic BP in humans followed either three or seven minutes inhalation of grapefruit, fennel, black pepper or tarragon oil vapors (Haze et al 2002). It is feasible that psychological factors may have caused these effects. However, intensive inhalation of grapefruit oil for 10 minutes raised BP in rats, due to the enhancement of sympathetic, and the inhibition of parasympathetic activity. An increase in BP of 10% was seen during the first 30 minutes, and this lasted for a further 60 minutes. There was no effect on rats with an inactivated sense of smell (Shen et al 2005, Tanida et al 2005). An unspecified amount of grapefruit oil was placed in the bottom of a beaker, and the rats had their nose placed in the beaker. Similar effects were seen from (+)-limonene inhalation.

Both psychological and pharmacological processes were considered responsible for increases in BP in humans following 30 minutes inhalation of (+)-limonene (systolic), (-)-limonene (systolic), (+)-carvone (diastolic), or (-)-carvone (systolic and diastolic). For example, fragrance-induced subjective ratings of increased alertness correlated significantly with increases in BP. Up to 500 mg of each substance was delivered through a breathing mask (Heuberger et al 2001).

Aromatic vapor inhalation does not always increase BP. After 10 minutes of inhaling 14.2 µg of vaporized cedrol, both systolic and diastolic pressures were reduced in healthy male and female Japanese volunteers, due to a reduction in sympathetic and an increase in parasympathetic activity (Dayawansa et al 2003).

**Mode of administration**

Perhaps not surprisingly, the mode of administration can make a significant difference to the resulting effect. Hypertension and convulsions can both be caused by acute methyl salicylate poisoning. However, topically applied methyl salicylate lowers BP through local effects (Dawson et al 2004, Futami 1984). In rats, (+)-limonene lowered BP on iv injection, but raised it on inhalation. There is no research that shows whether the use of single or blended
essential oils can lead to a significant increase in BP during an aromatherapy massage, but this seems unlikely since soft tissue massage itself reduces both systolic and diastolic pressure (Aourell et al 2005, McNamara et al 2003).

**Discussion**

Increases in BP have been recorded in both animals and humans on essential oil administration, but it is not known whether any of the data indicate a risk to people with hypertension. In all of the cited studies, animals and humans with normal blood pressure were tested. Most of the early (1940s) research was carried out by injecting dogs intravenously with moderately large doses of essential oil diluted in ethanol. In some of these, the precise quantity of essential oil used is not known, since only the amount of the total solution is given. Both increases and decreases in BP were recorded. Dermal administration of essential oil constituents can cause reductions in BP due to local effects. Conversely, most inhalation studies have reported BP increases.

Intravenous administration leads to a direct action on the vascular system, which is primarily due to calcium channel antagonism (Lahlou et al 2005). In inhalation studies, the effects are autonominically mediated, and in humans, psychological factors may come into play. In both rats and humans, elevations of BP were seen in conditions that reflect those of intentional and fairly intensive essential oil inhalation. Since soft-tissue massage reduces blood pressure, the only potential risks seem to be either from overdoses of certain convulsant essential oils, and from intensive (as distinct from incidental) inhalation.

**Conclusion**

Eucalyptus, camphor, pine, thyme and peppermint oils should be scratched from cautionary lists, as should cypress, ginger and rose. Hyssop and sage oils are only a risk in convulsant oral doses, and lower doses are very likely to be hypotensive. Therefore, they should not be contraindicated in hypertension. It is likely that rosemary oil follows the same pattern. Inhalation data suggest that essential oils presenting a risk include grapefruit, lemon, caraway, black pepper, fennel, tarragon and other oils high in carvone or limonene. However, in the human studies the increases were only slight.

Some essential oils may present a risk to some classes of hypertensive patient, in certain dose/route combinations. However, until we know more about where those risks lie, there is no case for contraindication of any essential oils.

© Robert Tisserand 2010
References


Battaglia S 1997 The complete guide to aromatherapy. The Perfect Potion, Virginia, Queensland


Caujolle F, Franck C 1945a Pharmacodynamic actions of clary sage and condiment sage. Comptes Rendues Société Biologique 139:1109-1110


Cazal R 1944 Contribution à l’étude de l’activité pharmacodynamique de quelques essences de labiées. These, Toulouse


Davis P 1999 Aromatherapy an A-Z. CW Daniel, Saffron Walden


Futami T 1984 [Actions and mechanisms of counterirritants on the muscular circulation]. Nippon Yakurigaku Zasshi 83:219-226


Lahlou S, Figueiredo AF, Magalhães PJ et al 2002 Cardiovascular effects of 1,8-cineole, a terpenoid oxide present in many plant essential oils, in normotensive rats. Canadian Journal of Physiology & Pharmacology 80:1125-1131


Tisserand R 1977 The art of aromatherapy. CW Daniel, Saffron Walden
