Neither lavender oil nor tea tree oil can be linked to breast growth in young boys

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Introduction

In 2007, a correlation was alleged between commercial products containing lavender and tea tree oils and breast growth in young boys. Three cases were seen in boys aged 4-7, who had all been using such products. In each case, the breast growth reduced to normal parameters within several months of ceasing to use the products. Subsequent laboratory testing showed that both essential oils had estrogen-like properties (Henley et al 2007).

In the report, no information was given about any of the product ingredients, and there is scant information on product use. No analysis was carried out to confirm or rule out the presence of essential oil constituents.

Case one
In the first case, "The patient's mother reported applying a "healing balm" containing lavender oil to his skin starting shortly before the initial presentation." No further details of the product or its use are given, but a healing balm sounds like something that might only be applied to a small area of skin. If so, then it is unlikely that any ingredient could have entered the boy's blood in sufficient concentration to cause gynecomastia within a short time period.

Case two
In the second case, a styling hair gel was applied to the hair and scalp every morning, along with regular use of a shampoo. Both tea tree oil and lavender oil are cited on the ingredient list of both products.

In a subsequent website report, it is claimed that the two hair products used in this case were manufactured by Paul Mitchell®, and that these were analyzed by a competitor. The shampoo was said to contain "very low concentrations" of tea tree oil, and the content in the hair gel was "virtually undetectable". Lavender oil concentration was not checked (Neustaedter 2007).

Dermal absorption of fragrance from shampoo application has been estimated to be 80 times less than that from body lotion (Cadby et al 2002) and tea tree oil constituents are poorly absorbed by human skin. In one study, only 3% of the essential oil volume, applied as a 20% concentration in ethanol, was absorbed in a 24 hour period (Cross and Roberts 2006). If the website report is reliable, considering that shampoo is a wash-off product, and that there was only a negligible amount of tea tree oil in the hair gel, tea tree

oil can be ruled out as a possible cause of this boy's gynecomastia. However, liberal use of a hair gel rich in lavender oil could result in moderate dermal absorption of lavender oil constituents (Cal 2006).

Case three

The third case involved "lavender-scented soap, and intermittent use of lavender-scented commercial skin lotions". This sounds as if there may not be very much natural lavender oil present. Since soap is a wash-off product, and use of the skin lotions is described as "intermittent", whether any meaningful absorption of lavender oil constituents took place at all seems doubtful.

As dermal absorption of soap fragrance is some 266 times less than that from body lotion, it is virtually impossible that the fragrance in a soap could be absorbed in sufficient quantity to cause any physiological effect (Cadby et al 2002).

Of great interest is the statement that, in this third case, a fraternal twin used the same skin lotions, but not the soap, and did not develop gynecomastia. It would be reasonable to assume that, since the soap could not be responsible for the effect, and since the twin used the lotions without any problem, the gynecomastia in this third case must have been due to some cause other than essential oils.

The *in vitro* testing

The *in vitro* evidence shows weak but definite endocrine disrupting effects for both lavender and tea tree oils.

The second case was the only one in which tea tree oil was involved. Tea tree oil was tested because it was deemed to be "chemically similar" to lavender oil. However, apart from the fact that both are essential oils, they have very little in common chemically.

The composition of the essential oils tested is not given, nor is any other information about them, apart from the supplier. Since they do not appear to be organically grown, biocide content is a possibility.

Discussion

It is unusual in such reports not to name the products suspected as being responsible for the effects in question. In the circumstances, it is also curious that the labeled ingredients were not cited. It is even more surprising that no attempt was made to ascertain, retrospectively, whether any constituents of lavender or tea tree oil were detectable, and if so at what concentrations. If the products are not named, no one else can test them either.

Subsequent research has confirmed that tea tree oil does show weak *in vitro* estrogenic action in MCF-7 cells (Nielsen 2008). However, none of the tea tree oil constituents that penetrate human skin (terpinen-4-ol, α-terpineol, 1,8-cineole) act as estrogens, either singly or in combination, in fact α-terpineol is anti-estrogenic (Cross et al 2008, Nielsen 2008, Reichling et al 2006). Tea tree oil is not a skin penetration enhancer, and in one study reduced the quantity of other substances (benzoic acid and methiocarb) crossing the dermal barrier (Nielsen and Nielsen 2006).

The two main lavender oil constituents, linalool and linalyl acetate, are absorbed by human skin (Jäger et al 1992). However, transcutaneous absorption from fragrances takes some time. The amount that could find its way into the blood from a wash-off product such as a shampoo or soap is negligible, because the time of skin contact is so short. Skin absorption from tea tree oil and lavender oil constituents is measured in hours rather than minutes, and in some instances even leave-on products result in minimal dermal penetration (Cal 2006, Reichling 2006).

No attempt was made to identify the constituent(s) responsible for the *in vitro* effect, but it is reasonable to expect that any hormonal action in an essential oil would be due to one or two constituents, or even contaminants. It is noteworthy that, while *in vitro* hormonal effects from essential oil constituents have been previously reported, these are generally very weak, and have been estimated as being at least 10,000 times less potent than 17β-estradiol (Howes et al 2002). Very weak activity is typical of estrogens from plant sources (Chadwick et al 2006).

There is no evidence that the effect seen *in vitro* would take place *in vivo*, and much more research would be needed before any such conclusion could be drawn. The report mentions that none of the boys had been exposed to any known endocrine disruptor, such as medications, oral contraceptives(!), marijuana or soy products. However, no mention is made of other known endocrine disruptors, including organochlorine pesticides, PCBs, polychlorinated dioxins, alkyl phenols, pthalates and parabens (Darbre 2006). Both pesticides and phthalates have been found in essential oils, and both phthalates and parabens are commonly found in cosmetic products.

Personal care products have been identified as contributing to phthalate body burden in adult men (Duty et al 2005); phthalates are commonly found in cosmetic products, and are potential hormone disruptors (Darbre 2006). DEHP, for example, has hormone disrupting effects in both male and female rats (Lovekamp-Swan and Davis 2003, Parks et al 2000), and high levels of several phthalates were found in the blood of 28 of 41 pre-pubertal girls (68%) with premature breast development compared to only 1 of 35 controls (3%) (Colón et al 2000).

It is, therefore, entirely possible that other ingredients or contaminants in the products caused the gynecomastia. Pesticides, PCBs and dioxins are found in the environment,

often in food, and it is also possible that a local surge of environmental hormone disruptors caused these cases in Colorado.

Conclusions

As the report states, breast growth in pre-pubertal boys is extremely uncommon, yet three cases were reported within a short period of time, and all in the same clinic. Considering that some 200 tonnes per annum are produced of both lavender oil and tea tree oil, that most of this goes into personal care products, and that very little of the evidence presented for these three cases is convincing, the initial press reports of caution were premature, as are the cautions now found on many websites.

Even if one or more of these cases was linked to product use, any connection with either lavender or tea tree oil is unproven. Other endocrine disrupting ingredients in the products could have played a role. Furthermore, we do not know what other factors, such as dietary or environmental, may have played a part.

The *in vitro* work reported by Henley et al (2007) does indicate a hormonal effect. However, this cannot be extrapolated to estimate actual human risk, especially without knowing more about the essential oil constituents causing the *in vitro* effects seen, the amounts being applied to the body, and their bioavailability.

No connection was established between the *in vitro* work and the three cases, and the evidence for tea tree oil having an effect on prepubertal gynecomastia is non-existent. Phytoestrogens generally have a very weak hormonal activity, and it is implausible that the amounts of essential oil that enter the body from product use would have a significant effect.

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