

## Odor, irritation, and the upper airway

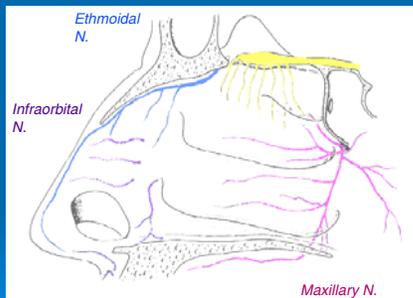


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## Chemosensory Modalities

- Smell (Cr. N. I)
- Trigeminal chemoreception (Cr. N. V)  
 (“common chemical sense”; “chemesthesis”)  
*Trigeminal N. distribution: Eyes, nose & throat*  
 (“Sensory irritation”)
- Taste (Cr. N’s. VII + IX)

## Innervation of the upper respiratory tract



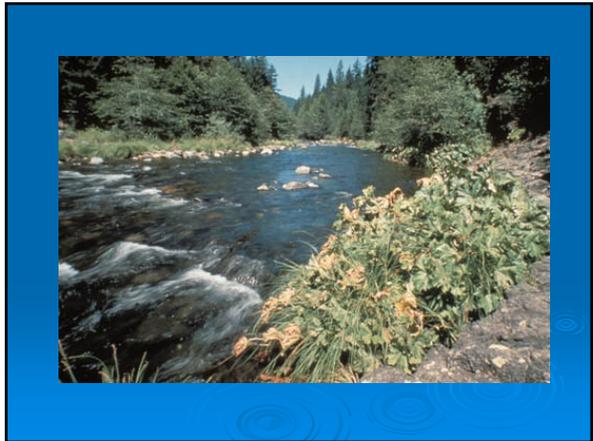
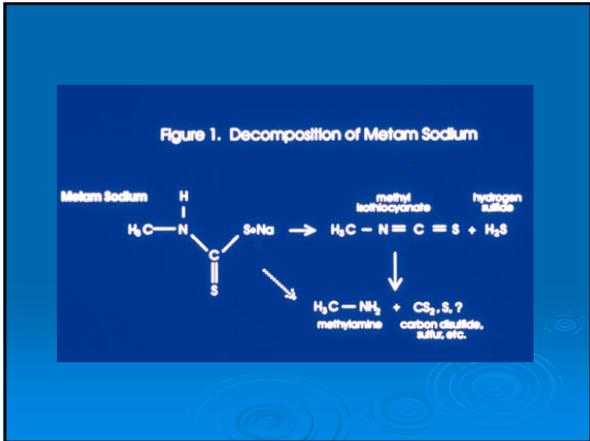
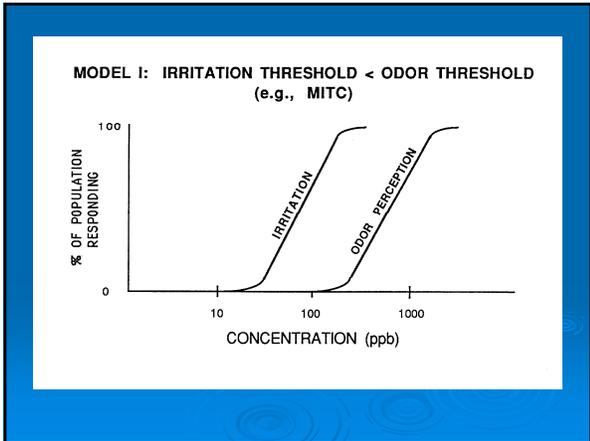
## Outline

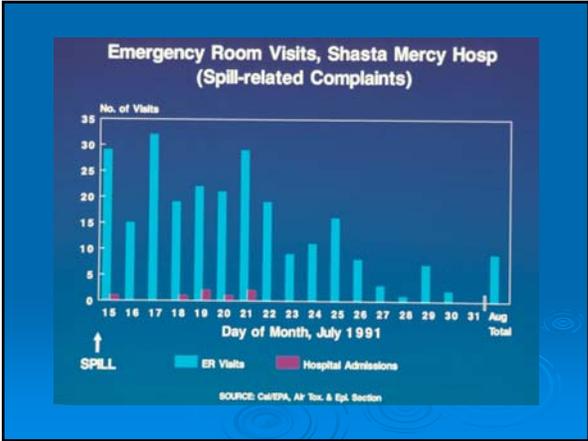
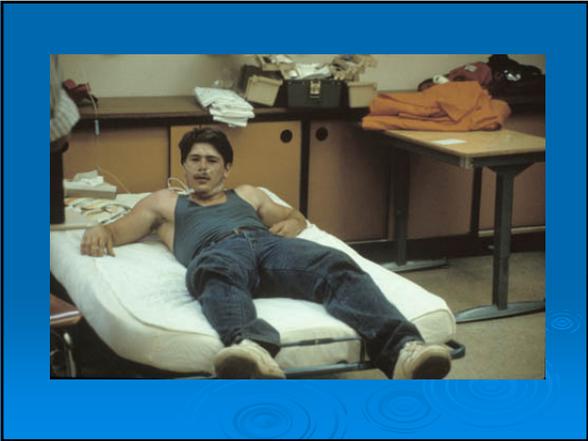
- Olfaction and trigeminal chemoreception are separate sensory modalities, but in everyday life give an integrated impression of the inspired atmosphere (e.g., “I smelled a *pungent odor.*”)
- Individual chemical agents, however vary in their relative olfactory and irritant potencies.
- The relative contribution of toxicologic (e.g., irritant) vs. non-toxicologic (i.e., odor-mediated) mechanisms varies by exposure scenario.

## Odor-related health effects

## Chemosensory Models

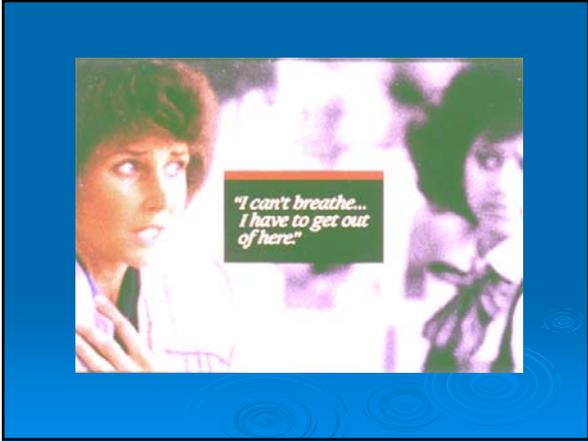
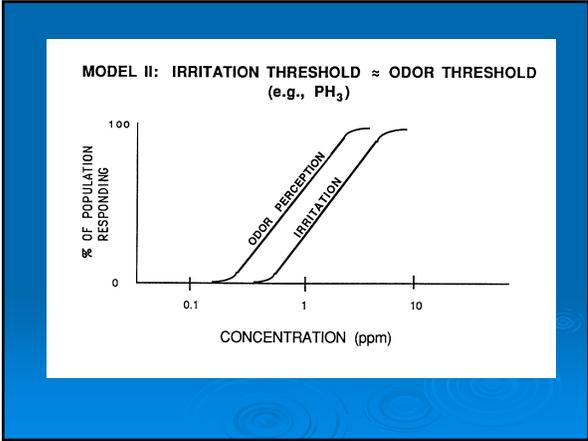
Model	Relative thresholds	Example	Comment
I	irritation < odor	MITC	Rare
II	odor < irritation	Phosphine	Many industrial chemicals
III	odor << irritation	H <sub>2</sub> S	Common air pollutants



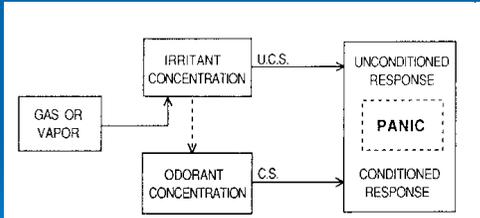


**Interpretation:**

- In the Sacramento River Metam sodium spill, chemical odors accompanied exposure. However, these odors, chiefly due to  $H_2S$ , served as a marker of exposure rather than a mediator of symptoms. The majority of symptoms were produced by a powerful irritant, methyl isothiocyanate, the sulfur analog of the chemical released at Bhopal, India. This chemical is unusual in that its irritant potency is > its odor potency.



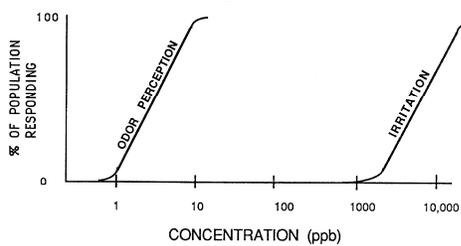
## Conditioning model of odor-related health effects



## Interpretation:

- Phosphine gas has an irritant threshold that is within one order-of-magnitude of its odor threshold.
- Levels of  $\text{PH}_3$  that were tolerated by this worker prior to the overexposure incident were no longer tolerated after the incident.
- The mechanism whereby the odor of  $\text{PH}_3$  triggered hyperventilation / panic symptoms is thought to be classical (Pavlovian) conditioning.

MODEL III: IRRITATION THRESHOLD > ODOR THRESHOLD  
(e.g.,  $\text{H}_2\text{S}$  & Mercaptans)

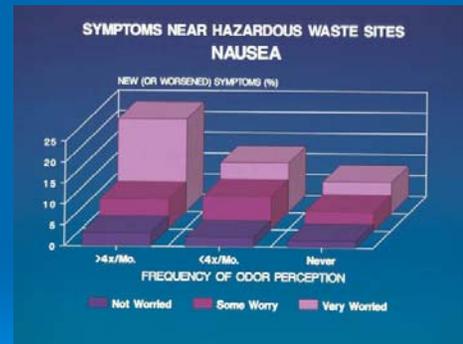
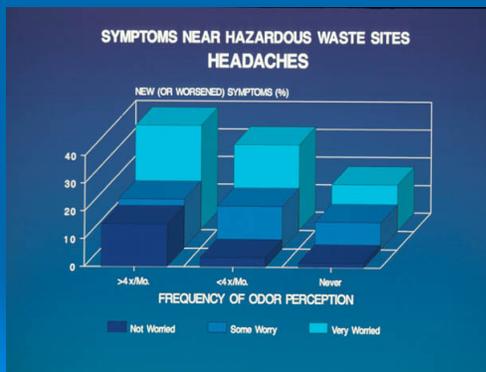




### “Symptom prevalence and odor-worry interaction near hazardous waste sites”

Shusterman, Lipscomb, Neutra, Satin.  
 Environ Health Perspect 1991

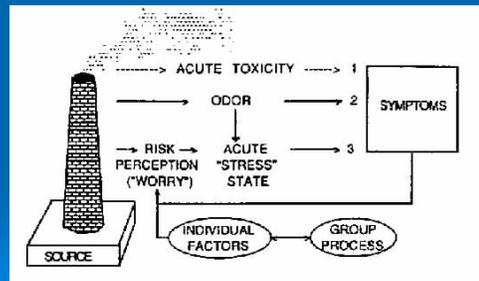
- Meta-analysis of data from 3 symptom prevalence surveys conducted near hazardous waste sites in Southern California
- Respondents asked re: incidence of selected symptoms (headache, nausea, eye and throat irritation)
- Respondents asked “How often do you smell unusual environmental odors?”
- Respondents asked “How worried are you about the quality of your neighborhood environment?”



### Interpretation:

- Frequency of self-reported “unusual environmental odors” predicted symptom reporting.
- Degree of self-reported “environmental worry” also predicted symptom reporting.
- The relationship of these two variables and symptom reporting was *supra-additive*.
- One explanation is that environmental worry *potentiates* odor-related symptom reporting.

### Interactive model of odor-related health effects



## Summary:

- Upper respiratory tract innervation includes *olfaction* and *chemesthesis* (sensory irritation).
- Individual airborne chemical agents differ in their relative *odorant* and *irritant* potencies.
- Odors can *trigger symptoms* by a variety of non-toxicological mechanisms.
- *Cognitive mindset* (risk perception, expectations, prior experience) affect both primary (sensory) and secondary (symptomatic) responses to air pollutants at low (odorant) - as opposed to high (irritant) – concentrations.