« La Diossina…..!!!!!?????»

ArcipelagoMilano
informazione - politica - cultura

5 gennaio 2019

QUANTI SONO I CAPANNONI PRONTI A BRUCIARE IN LOMBARDIA?

L'inquinamento e il controllo del territorio

di Valeria Fieramonte


Domenica 18 luglio 1976 CORRIERE DELLA SERA

CORRIERE DELLA METROPOLI

UNA ORDINANZA DEL SINDACO VIETA DI INGERIRE E TOCCARE I PRODOTTI ORTOFRUTTICOLI DELLA ZONA

Nube tossica: le immagini del dramma di Seveso

Oggi comincia l'opera di inceneri mento di tutti gli elementi contaminati nell'area investita, predisposta dalle autorità per evitare ulteriori danni - Consulto all'ospedale di Mariano Comense per stabilire la cura più adatta a guarire le ustioni dei bambini

Nel '76, quando è esplosa la Givaudan, liberando quantitativi di diossina che hanno costretto a evacuare parte della popolazione di Seveso e dintorni, ero una giovane cronista di una radio. Già conoscevo Laura Conti, che su Seveso ha condotto una campagna politica di grande impatto e bravura. (La direttiva

Il recente, siamo nell'ottobre del 2018, incendio della Bovisasca e di Quarto Oggiaro, a Milano, va ad aggiungersi agli altri 150 incendi nell'ultimo anno in Lombardia, di cui una ventina gravissimi (es. incendio nei pressi di Pavia, ora sarebbero stati individuati gli autori, dopo quasi un anno, e questo della Bovisasca). Ma negli ultimi anni ne sono stati contati molti più di 300, (per la precisione 343, che non è una cifra precisa perché ogni giorno, specie nei depositi piccoli, brucia qualche plastica).

La diossina, che in sostanza è il prodotto della combustione di petrolio più cloro, è una sostanza tossica liberata dalla maggior parte delle plastiche, (ovviamente non quelle biodegradabili), è mutagena e cancerogena: alcuni tecnici che erano allora in fabbrica sono morti di tumore al fegato, la principale malformazione è il labbro leporino, ma se l'intossicazione è grave, in genere ci sono aborti spontanei (per fortuna)…….

???

NO

Non so chi siano i pazzi criminali che hanno fatto stoccare la plastica proprio nella Pianura Padana, una delle zone con l'aria più inquinata del mondo. I tempi pachidermici della giustizia civile e penale non sono compatibili con l'imminente rischio di una catastrofe ambientale: **la diossina che va ora accumulandosi, anche se non è ultratossica come quella di Seveso al triclorofenolo, rischia di devastare l'agricoltura della pianura padana, ovvero del motore economico italiano.**

……

Per quanto riguarda specificamente la nuvola della Bovisasca, il secondo giorno dell'incendio la diossina nell'aria, dove in genere si disperde più velocemente, era di ben **7 picogrammi**! Non 0,7 com'è stato pare scritto da alcuni giornali. Come per tutte le sostanze che sono anche mutagene, non c'è valore di soglia: sopra gli 0,3 picogrammi vuol dire però che occorre iniziare a preoccuparsi, perché ci sono focolai attivi. Il valore di 0,3 lo si trova sempre, da Seveso in poi, perché, nella terra non coltivata è nei primi 20cm. Nella terra coltivata è più sotto per via delle vangature. Perciò a essere interessati non sono i polmoni ma il fegato, che fa da filtro e sviluppa tumori. C' è il sospetto che in alcuni capannoni sia bruciato anche l'amianto. Come mai non c'è valore-soglia per le sostanze mutagene?

Contaminanti considerati

microinquinanti organoclorurati **“diossina simili”**

DIOSSINA ?

Importanza del linguaggio

ai fini di una corretta comunicazione

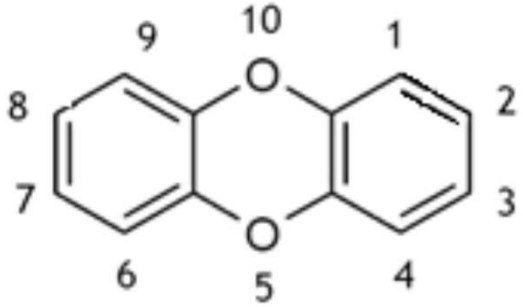
PolyChloroDibenzo-*p*-Dioxins, PCDD
PolyChloroDibezoFurans, PCDF

- **Microinquinanti organo-clorurati costituiti da due anelli benzenici e uniti da ponti ossigeno;**
- **Elevata tossicità dei congeneri 2,3,7,8 sostituiti, in particolare: 2,3,7,8 -TeCDD e 2,3,4,7,8-PeCDF;**
- **Persistenti in ambiente (POPs), liposolubili, poco volatili.**

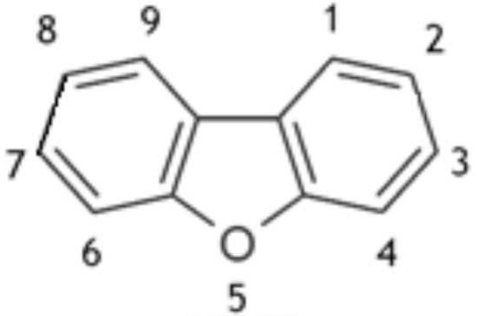
IN TOTALE:

75 congeneri PCDD

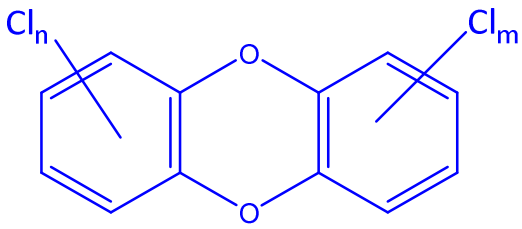
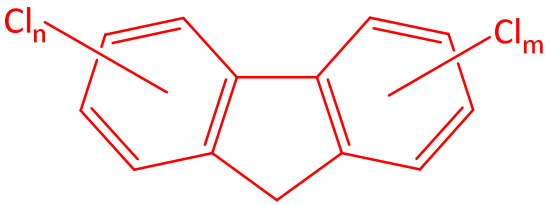
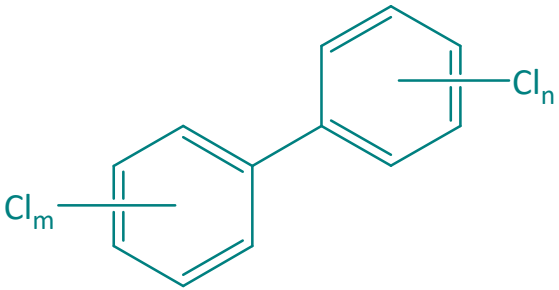
135 congeneri PCDF



PCDD



PCDF

		Isomeri	Isomeri tossici	%
PCDD		75	7	9.3
PCDF		135	10	7.4
PCB		209	14	6.7
		419	31	7.4

How much is too much?

How is toxicity measured?

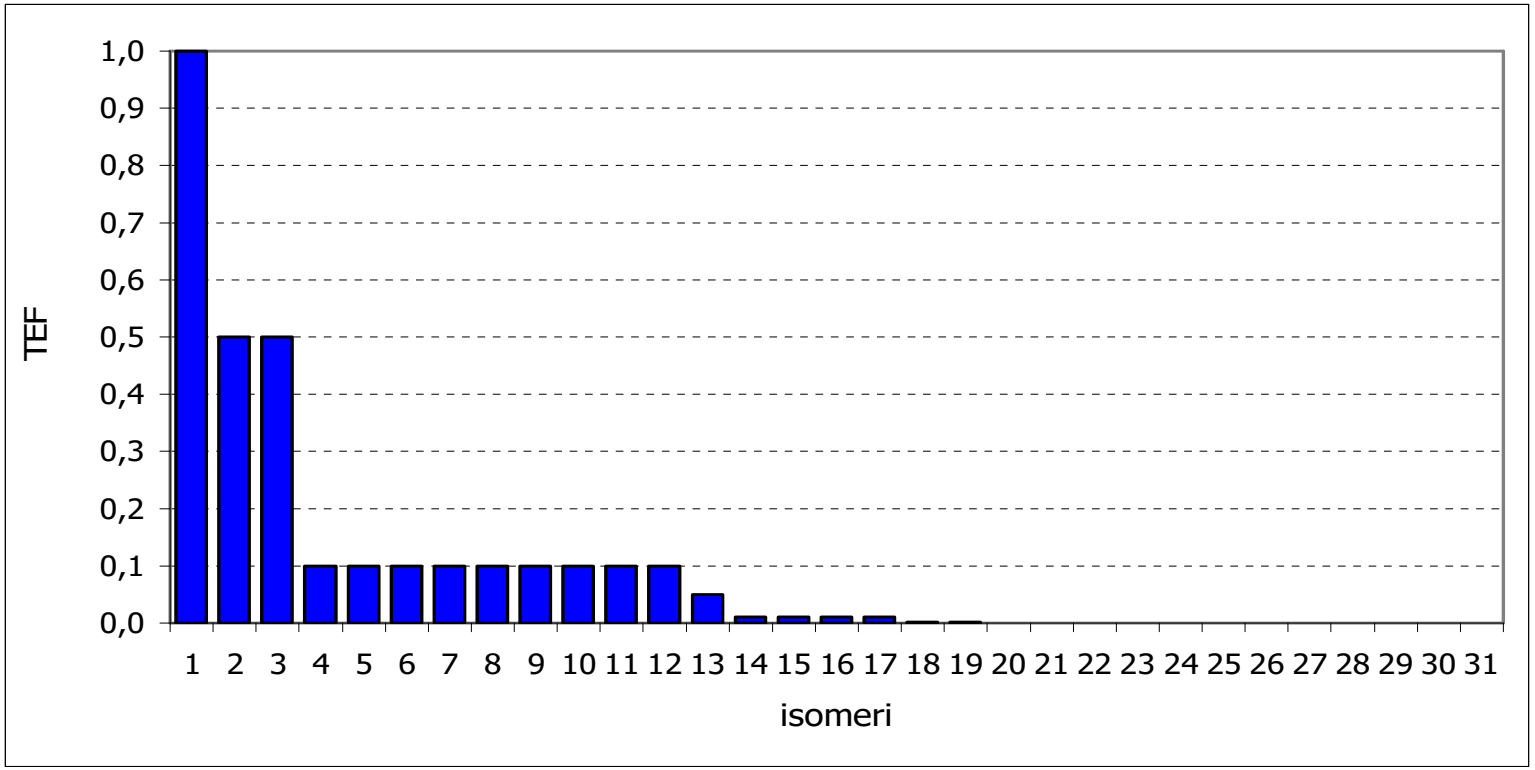
- Dioxin toxic equivalency factor (TEF) is a number assigned based on potency compared to the most toxic dioxin, TCDD.
- TEF x Concentration = concentration equivalent to TCDD (toxic equivalent, TE)
- Sum TEF of a mixture

  Add the partial equivalents of all compounds in the mixture.
- Important to note that these are based assumptions

  used "…as a proxy of the total toxic amount of dioxin like compounds" (Linden et al. 2010).

Valutazione effetti su salute

Solo 31 dei 419 isomeri (7,4%) hanno effetti tossicologici riconosciuti



Tossicità di miscele

I composti “diossina simili” sono presenti
nell’ambiente e nei campioni biologici come miscela complessa.
Per consentire la valutazione del rischio e il controllo dell’esposizione (con valori limite) è stato introdotto il concetto di “Tossicità Equivalente” (TEQ).

$$\mathbf{TEQ = \sum ([PCDD_i] \times TEF_i) + \sum ([PCDF_i] \times TEF_i) + \sum (CPCB_i \times TEF_i)}$$

In emissione espressi come ng_{TEQ}/Nm^3

Dioxin poisoning-Who is susceptible?

- **Fetus:** the developing fetus is most susceptible. Exposure occurs through food and other materials that the mother ingests or that she is exposed to.

- **Industrial workers:** industrial workers are exposed to dioxins on a daily basis (i.e. hazardous material on the job, factory waste, etc.)

- **Populations with high-fish/shellfish, high-animal fat diet:** dioxins are dissolved in the fat of animals and marine animals in the environment.

- **General population:** through background exposure (Background exposure tends to be relatively low, except in Viet Nam, where environmental levels remain extremely high.)

What are the effects of Dioxin Exposure?

- Dioxin poisoning results in a spectrum of disorders
- Chloracne is the hallmark of dioxin exposure in humans
  - Chlorance can persist for years, or even decades

What are the effects?

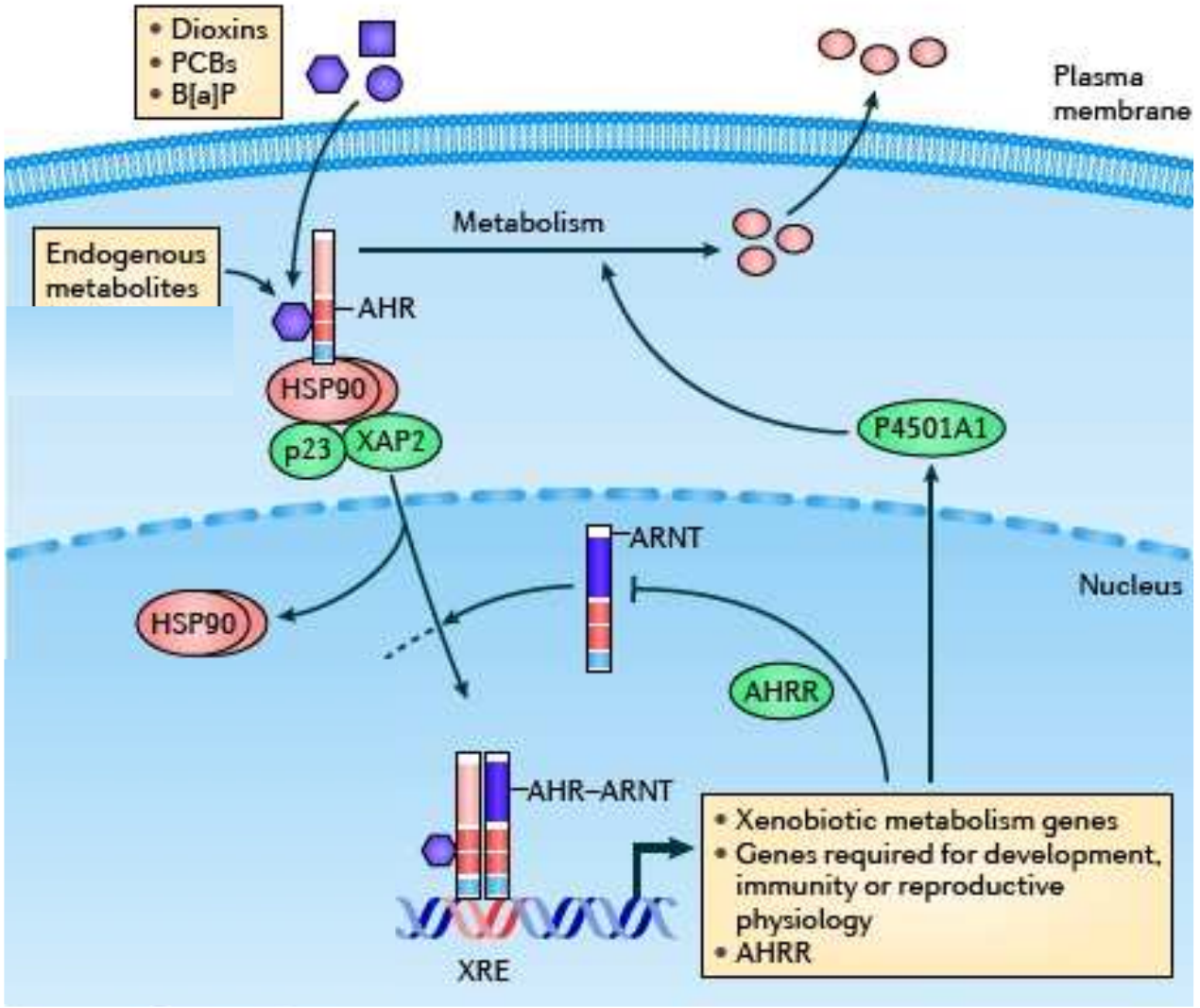
- **Acute symptoms/short term**
  - skin lesions
  - skin darkening
  - liver dysfunction
- **Chronic symptoms/long term**
  - diabetes
  - cancer
  - birth defects- physical deformities, mental disabilities, etc.
  - infertility
  - other disabilities
- **Effects on babies and/or pregnant women**
  - impairs nervous system development
  - impairs normal development
  - dissolves in breast milk
- **Effects on children/adolescents**
  - impaired immune function → cancer
  - impaired endocrine function → diabetes, infertility
  - impaired reproduction

Mechanism of Action

- Why are dioxins toxic?
  - Interactions with the ligand-mediated transcription factor aryl hydrocarbon (AhR)
    - Dioxins are ligands of AhR

Classical mechanism of action

HSP90: heat shock protein 90
XAP2: HBV X-associated protein 2
ARNT: AhR nuclear translocator
AHRR: AHR Repressor
XRE: xenobiotic response element



Bersten et al., Nat Rev Cancer 2013, 13, 827

HISTORY OF DIOXIN PRODUCTION AND EXPOSURES

J Environ Sci Health C Environ Carcinog Ecotoxicol Rev. 2009 October ; 27(4): 197–211.

The history of anthropogenic **dioxin** production and dioxin poisoning is nearly 200-years old and persists today.

The earliest evidence of man-made dioxin molecules comes from a German chemical production plant in Lampertheim, South Hesse that was manufacturing washing soda (sodium carbonate, also known as sal soda or soda ash) by the *LeBlanc* process as early as 1827, and until at least around 1890, when it was replaced with chloralkali electrolysis – both of which processes generated dioxin.

It was not until the 1980s, when a playground and a facility intended for children's use were slated to be built on the site where the plant had stood, that the extensive dioxin soil contamination was identified. It was many decades after the polluting of this site began, that chloracne was first characterized in 1897. This persistent cystic and hyperkeratotic skin condition, first identified in German industrial workers, remains a hallmark of dioxin exposure.

Between 1962 and 1970 **Agent Orange**, the American military code name for an herbicide containing 2,4,5-T and contaminated again with TCDD – similar to the contaminated 2,4,5-T in the 1949 Monsanto explosion – was used as a defoliant in Vietnam to reduce enemy ground cover, as part of Operation Ranch Hand. Recent studies of the Ranch Hand cohort have revealed that American military exposures to Agent Orange were associated with an increased risk of diabetes and an increased risk of multiple cancers, with increased duration of potential exposure conditions.

In 1968 in **Kyushu, Japan**, a rice bran oil company's supplies became contaminated with PCBs and PCDFs, and the contaminated oil was sold and fed to livestock and humans, resulting in the deaths of hundreds of thousands of birds. In humans, skin lesions, fatigue, and altered reproductive and immunologic function were symptoms of what was referred to as "Yusho" (literally, "oil") disease, and developmental delays were also observed in children.

Sadly, this incident was essentially repeated in **Taiwan** in 1979, but was referred to in Chinese as "Yucheng" disease, also meaning "oil." In this event, the association between gestational or lactational exposure was more clearly associated with impaired cognitive development and behavioral problems, and recent studies have also demonstrated persistent effects, with respect to altered reproduction parameters post-pubertally among the exposed males.

Such events were not isolated to Asia. In the interim between the Yusho and Yucheng poisonings, in 1971 an entire town in the United States was exposed to high levels of dioxins when contaminated waste oil was spread on the dirt roads of **Times Beach, Missouri**, in order to control dust. In the very same year, TCDD was identified as a teratogen.

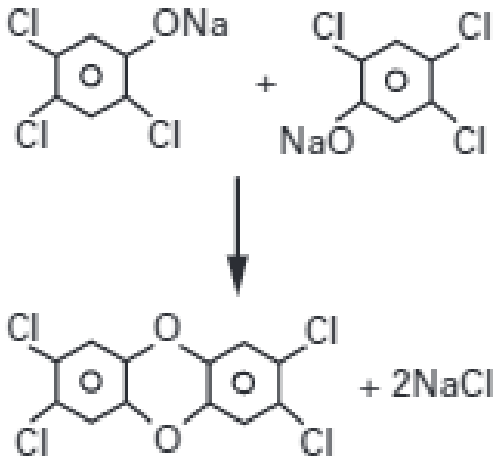
Ultimately, all properties in Times Beach were bought out by the US EPA for a total of $32M in 1983, the inhabitants were relocated, the town was demolished, and 265,000 tons of regional soil were incinerated. This paved the way for the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980, or the Superfund law, as it is more commonly known.

In **Binghamton, NY** in 1981, a PCB dielectric fluid-filled transformer caught fire in the basement of the Binghamton State Office Building. The fire deposited an oily soot throughout the 18-story building, most notably on the horizontal surfaces, where PCDDs and PCDFs were deposited at levels that varied within the building, but were measured as high as 1200 ppm. As a result of the extensive contamination throughout the structure, millions of dollars were spent on cleaning and remediation, and the building was shuttered for 13 years

In 1976, in **Seveso, Italy**, an explosion occurred at an Italian chemical plant producing 2,4,5-trichlorophenol, an intermediate in 2,4,5-T synthesis. Because of the nature of the uncontrolled reaction that produced the explosion, not only was TCDD released into the outside environment when the facility was breached, but its levels were far higher than the normal range of 1 ppm for contamination of 2,4,5-T, and may have approached 100 ppm. Within several weeks of the accident, some of the exposed community members exhibited skin lesions consistent with chloracne. In the years that followed, continuing studies of the exposed population supported the potential for TCDD to act as a carcinogen in humans and to increase risk for diabetes, adverse cardiovascular effects, and altered endocrine function.

Seveso - ICMESA

Produzione di triclorofenolo



2,3,7,8-TCDD

B. Eskenazia, M. Warner, P. Brambilla, S. Signorini, J. Amesa, P. Mocarelli, "The Seveso accident: A look at 40 years of health research and beyond", Environment International ***121*** *(2018) 71-84.*

Most recent in people's memories, of course, is the dioxin **poisoning of Viktor Yushchenko in 2004.** The then-candidate and now president of Ukraine may have been intentionally poisoned with TCCD, as part of measures to weaken his political influence and potentially remove him from the campaign. Two months prior to the presidential election, Yushchenko was hospitalized for pancreatitis, which was followed by profound facial acne and edema. The diagnosis of chloracne was confirmed when dioxin blood levels were found to be three orders of magnitude above average. His was not the first case of intentional dioxin poisoning, however. In Vienna in 1997, five people working in secretarial positions at a textile institute were seemingly poisoned with extremely high levels of dioxin, one of whom exhibited the highest serum dioxin levels ever measured in an individual -- 144,000 pg TCDD/g blood fat.

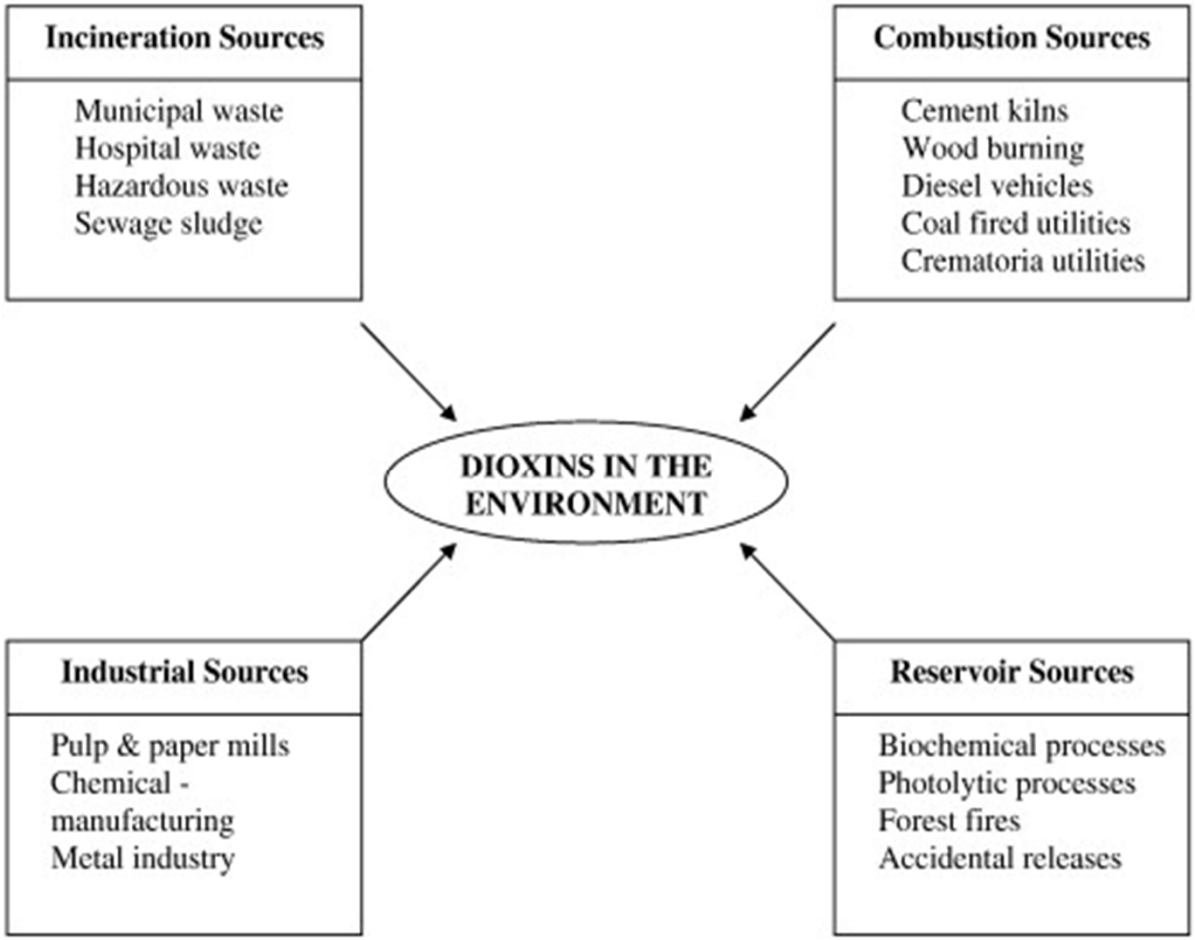
Finally, as 2008 drew to a close, all Irish pork products were removed from international shelves due to the known contamination of these animals resulting from ingestion of dioxin- contaminated feed supplies. Disturbingly, this event was essentially a repeat of the Belgian contamination nearly ten years prior

What are the sources of Dioxins?

- Anthropogenic in origin

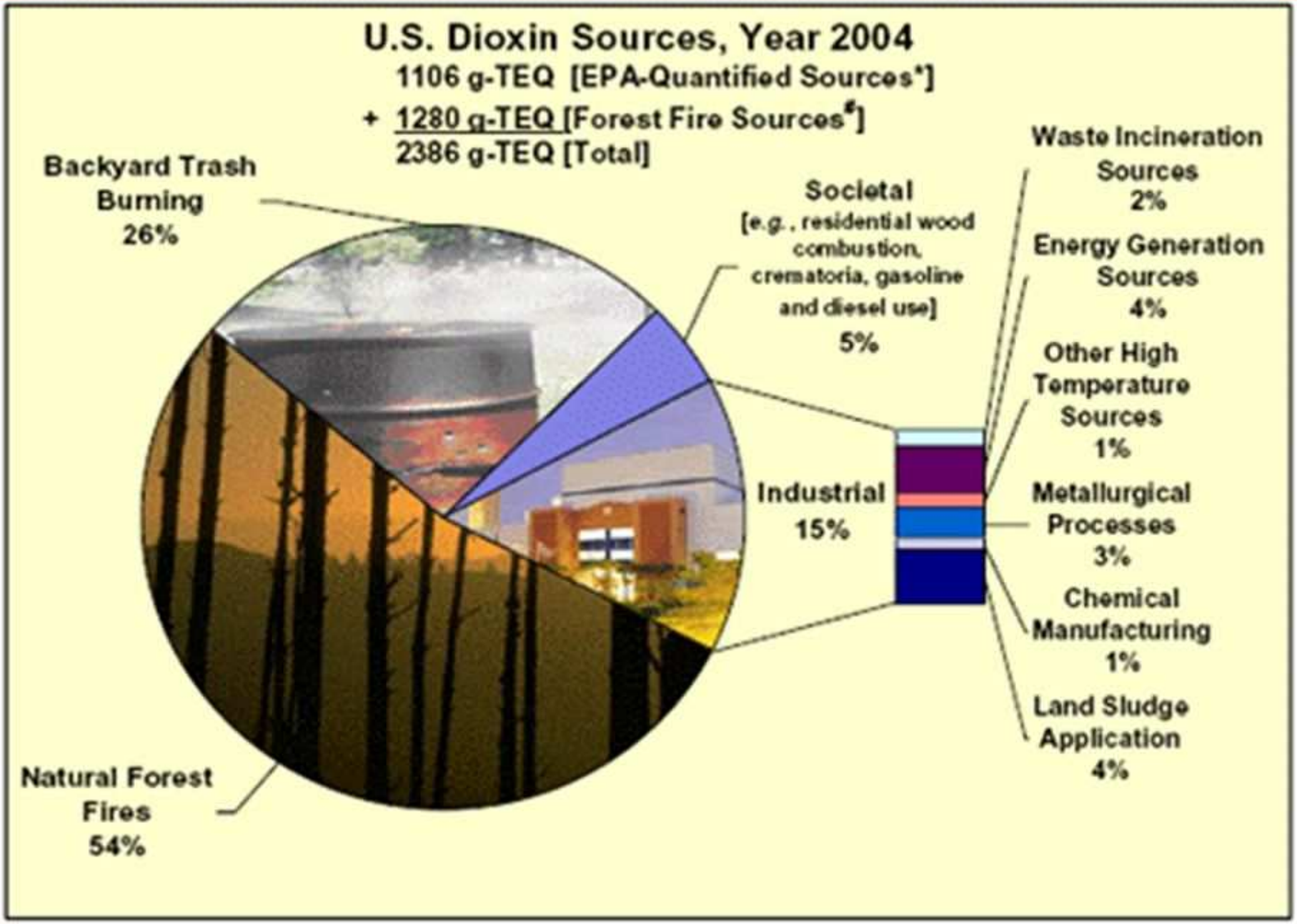
- "The only natural sources of dioxins are forest fires and volcano activities. Most are formed and released as by-products of human activities".

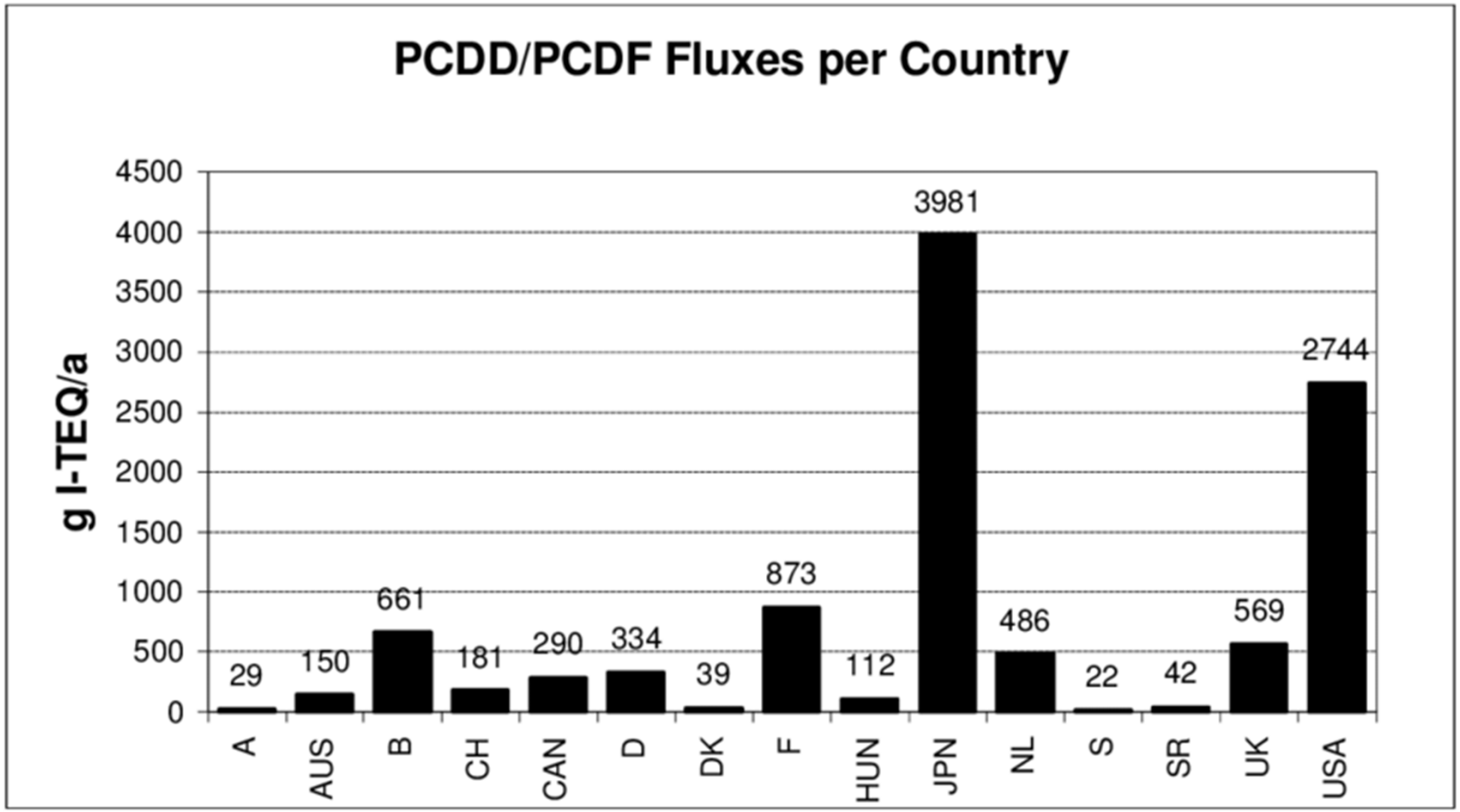
What are the sources of Dioxins?



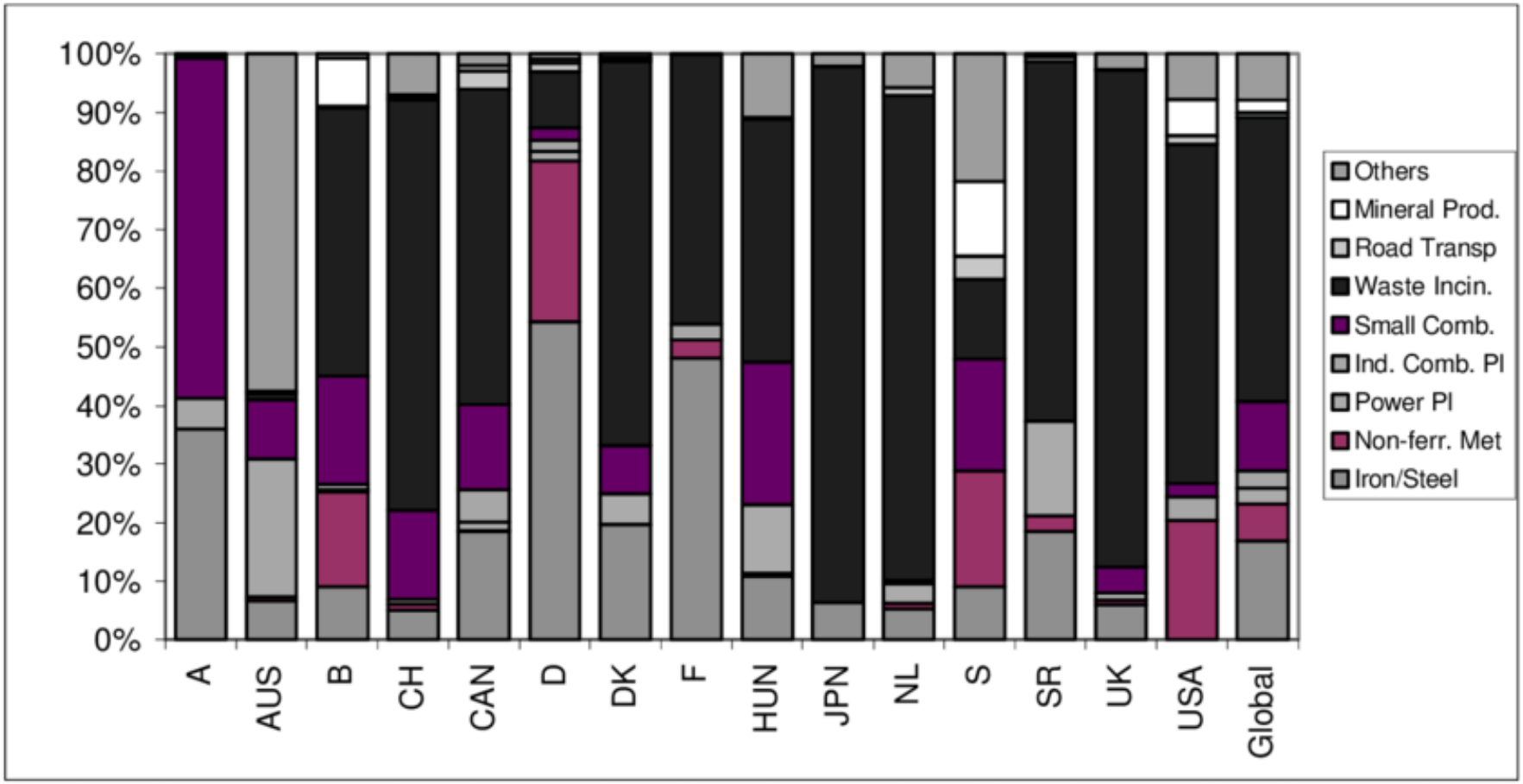
Dioxins are also found in herbicides and pesticides.

Combustion is major source of some dioxins





PCDD/PCDF annual fluxes into air (g I-TEQ/a); reference year 1995. Total emissions from known sources = 10,500 g I-TEQ/a



Percentage contribution per sector and country to the overall PCDD/PCDF air emission inventory; reference year 1995

Table 44. European PCDD/PCDF emission inventory per sector (concentrations in g I-TEQ/a) [110]

Sector	1995 (min-max)	2000 (min-max)	Change
Power plants – fossil fuel	59–122	55–72	–30%
Resident. comb. – wood	544–989	532–971	– 2%
Resident. comb. – coal, lignite	92–408	86–370	– 9%
Industrial boilers, stationary engines	32–83	34–81	0%
Sinter plants	671–864	447–554	–35%
Sec. zinc production	242–245	22–25	–90%
Sec. copper production	31–33	15–17	–50%
Sec. aluminum production	41–50	13–49	– 2%
Cement	14–50	13–49	– 2%
Metal cable reclamation	42–52	40–50	– 3%
Electric arc furnace steel plants	115–162	120–153	– 1%
Non-ferrous metal foundries	36–78	40–74	0%
Sintering of spec. materials, drossing facilities	115–200	1–86	–72%
Preservation of wood	145–388	131–349	–10%
Road transport	57–138	37–82	–39%
MSWI (legal)	973–1,213	412–506	–58%
MSWI (illegal)	129–221	126–200	– 7%
Incineration of industrial waste	149–183	131–166	–10%
Incineration of hospital waste	133–530	96–392	–27%
Crematoria	11–46	9–19	–51%
Total	3,685–6,470	2,435–4,660	–30%
– Industrial sources	2,793–4,165	1,589–2,516	–41%
– Non-industrial sources	892–2,305	846–2,144	– 6%

How are we exposed to dioxins?

- **Industrial waste:** (factories)
  - improper disposal.

- **Pesticides/herbicides:** (Agent Orange)
  - 11.4 million gallons dropped in Viet Nam between 1961 and 1970 at up to 55 times the suggested concentration.

- **Exposed through:**
  - soil, sediments, food (dairy, meat, fish, shellfish), breast milk, and passage through the placental barrier.

- **How can exposure be limited?**
  - Limit animal fat intake (especially pregnant women and nursing mothers)

Chemosphere No. 8, pp 455 - 459, 1977. Pergamon Press. Printed in Great Britain.

CHLORODIBENZO-p-DIOXINS AND CHLORODIBENZOFURANS ARE TRACE COMPONENTS OF FLY ASH AND FLUE GAS OF SOME MUNICIPAL INCINERATORS IN THE NETHERLANDS.

K. Olie, P.L. Vermeulen, O. Hutzinger[×]

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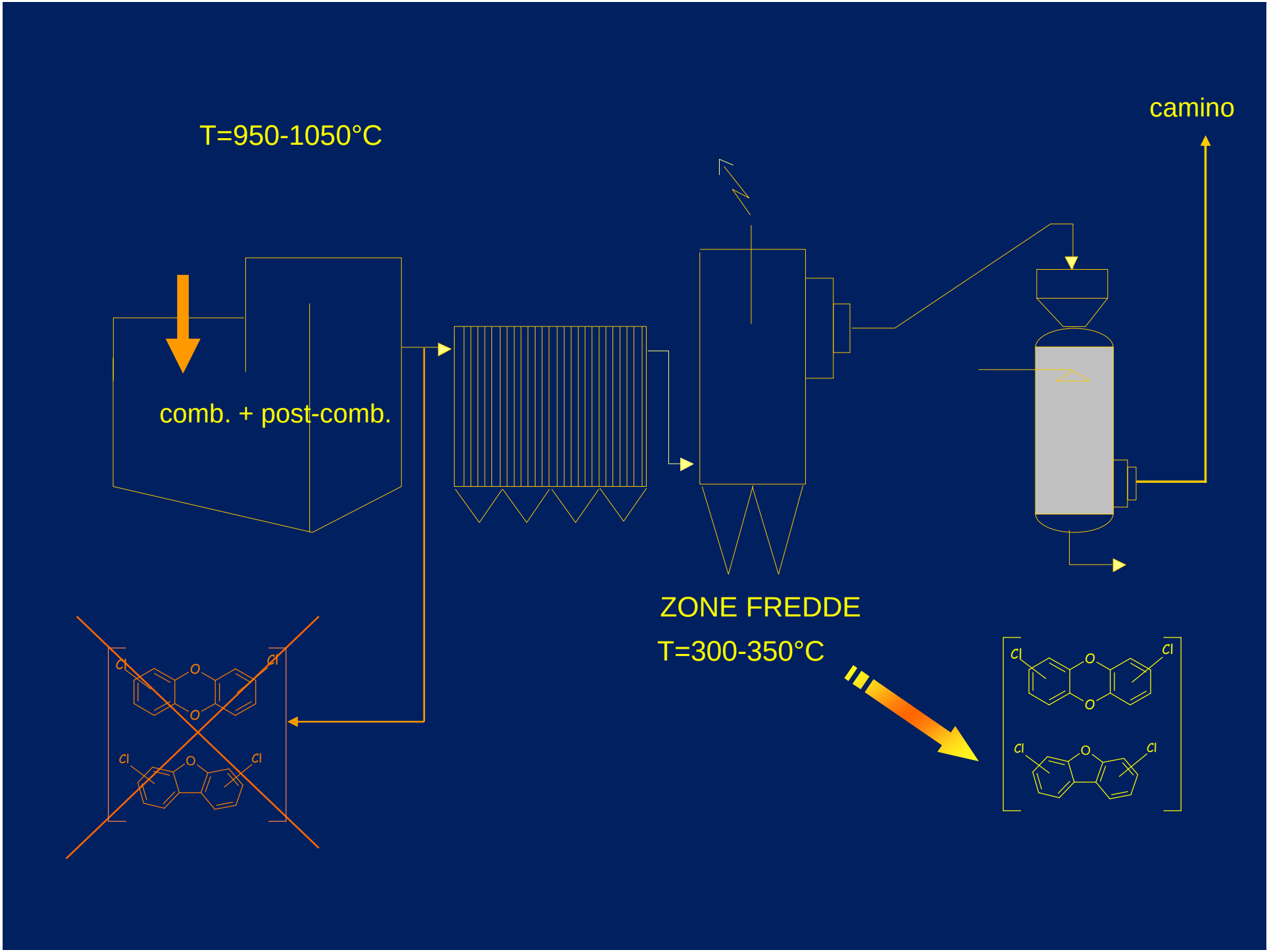
(Received in The Netherlands 1st July 1977; accepted for publication 14 July 1977)

Although no quantitative data on the release of these toxic materials into the environment via municipal incinerators are available at the moment the following generalisation from our qualitative measurement can be made:

(i) The amount of chlorodibenzodioxins and chlorodibenzofurans entering the atmosphere via flue gases is probably quite small. However, due to the extreme toxicity of some of the components and the fact that most incineration plants are located in densely populated areas extensive monitoring of such facilities might be advisable.

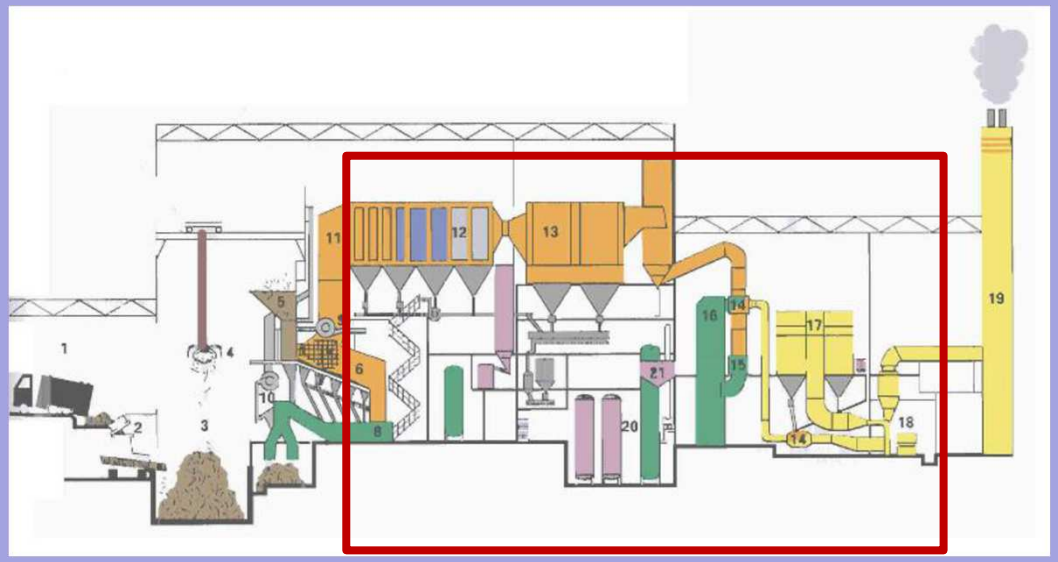
(ii) Since the origin of chlorodibenzodioxins and chlorodibenzofurans in fly ash and flue gases of municipal incinerators is unknown and since conditions of high temperature are most likely important, fly ash and flue gases of other installations from industry and energy production should be investigated. In the past analysis was mainly for toxic metals[4,5] and well-known flue gas components such as carbon monoxide, hydrogen chloride and sulphur dioxide and more recently polycyclic hydrocarbons[6].

(iii) Municipal incinerators and other combustion processes may be a source of some of the organochlorine compounds in the environment. The origin of highly chlorinated benzenes in the environment for example has not been adequately explained and these compounds have been found in all our samples of another research group[7].


T=950-1050°C
camino
comb. + post-comb.
ZONE FREDDE
T=300-350°C

SINTESI PCDD E PCDF NEI TERMOVALORIZZATORI

I PCDD/F eventualmente presenti nei rifiuti vengono distrutti nella fase di combustione

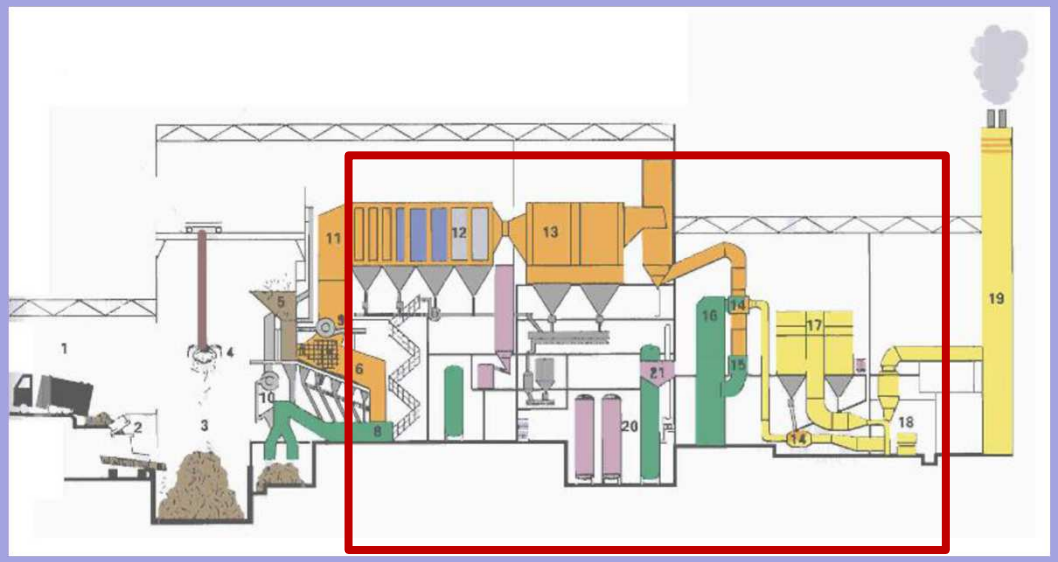


RECUPERO CALORE E TRATTAMENTO FUMI

Al camino:

PCDD/F input < PCDD/F output

SINTESI PCDD E PCDF NEI TERMOVALORIZZATORI



ZONE FREDDE
(200-400°C)

SINTESI PCDD/F

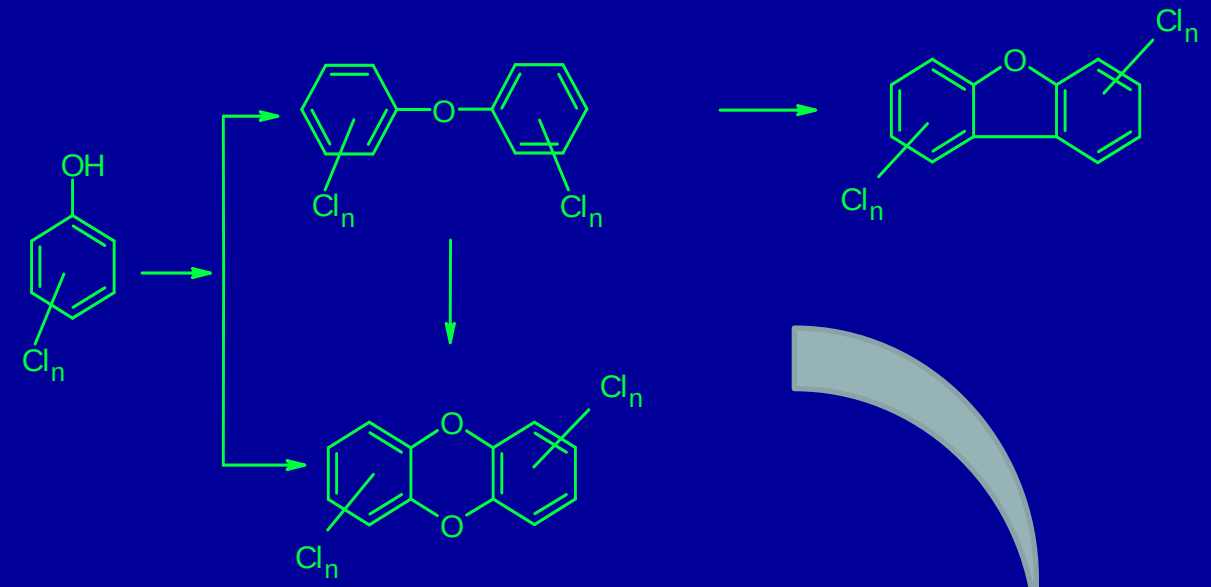
- Fase omogenea (500-800°C)
- Fase eterogenea (200-400°C)
  - Sintesi da precursori
  - SINTESI DE NOVO

PCDD/F si riformano nelle zone a bassa temperatura dell'impianto, sulla superficie delle *fly ash*

SINTESI PCDD E PCDF NEI TERMOVALORIZZATORI

Sintesi da precursori

Policlorobenzeni, policlorofenoli

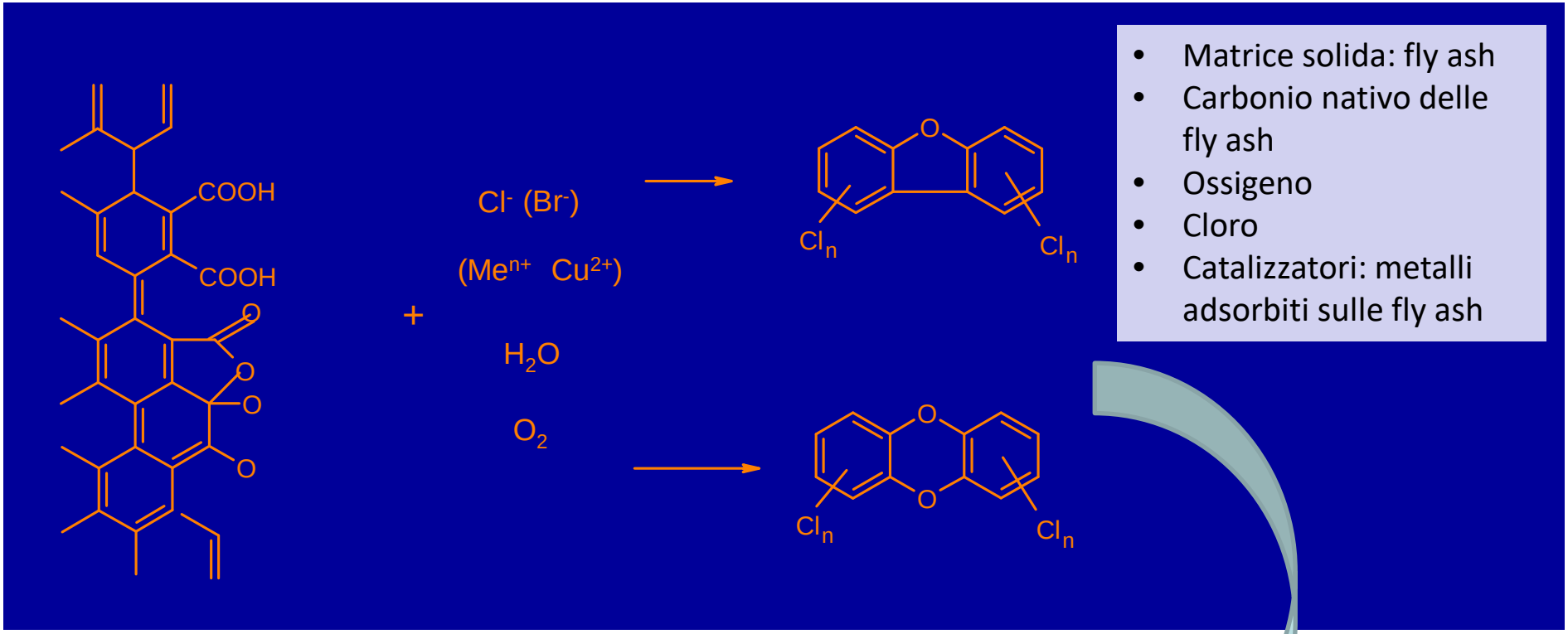


fly ash può agire da catalizzatore

PCDF/PCDD < 1

SINTESI PCDD E PCDF NEI TERMOVALORIZZATORI

SINTESI DE NOVO



- Matrice solida: fly ash
- Carbonio nativo delle fly ash
- Ossigeno
- Cloro
- Catalizzatori: metalli adsorbiti sulle fly ash

PCDF/PCDD > 1

Migliore Tecnologia Disponibile (Best Available Technology, BAT):

CARBONI ATTIVI e FILTRI A MANICHE *(END OF PIPE STRATEGY)*

Tecnologie Ambientalmente Sostenibili (Environmentally Sound Technologies, EST)

Punti chiave

- ✓ utilizzare le risorse in modo sostenibile
- ✓ dare adeguata protezione all'uomo e all'ambiente
- ✓ avere meno emissioni inquinanti
- ✓ riciclare la maggior parte dei sottoprodotti
- ✓ consentire il trattamento e lo smaltimento dei reflui finali
- ✓ sviluppare processi ambientalmente più accettabili

> "Sustainable development is development that meets the needs of the present without compromising the ability of future generations to meet their own needs"
>
> (Brundtland, 1987)

OBIETTIVO

Studiare a scala di laboratorio la cinetica delle reazioni di formazione e distruzione di PCDD/F

Identificare delle strategie che permettano di minimizzare le concentrazioni di PCDD e PCDF nelle emissioni generate da processi termici industriali

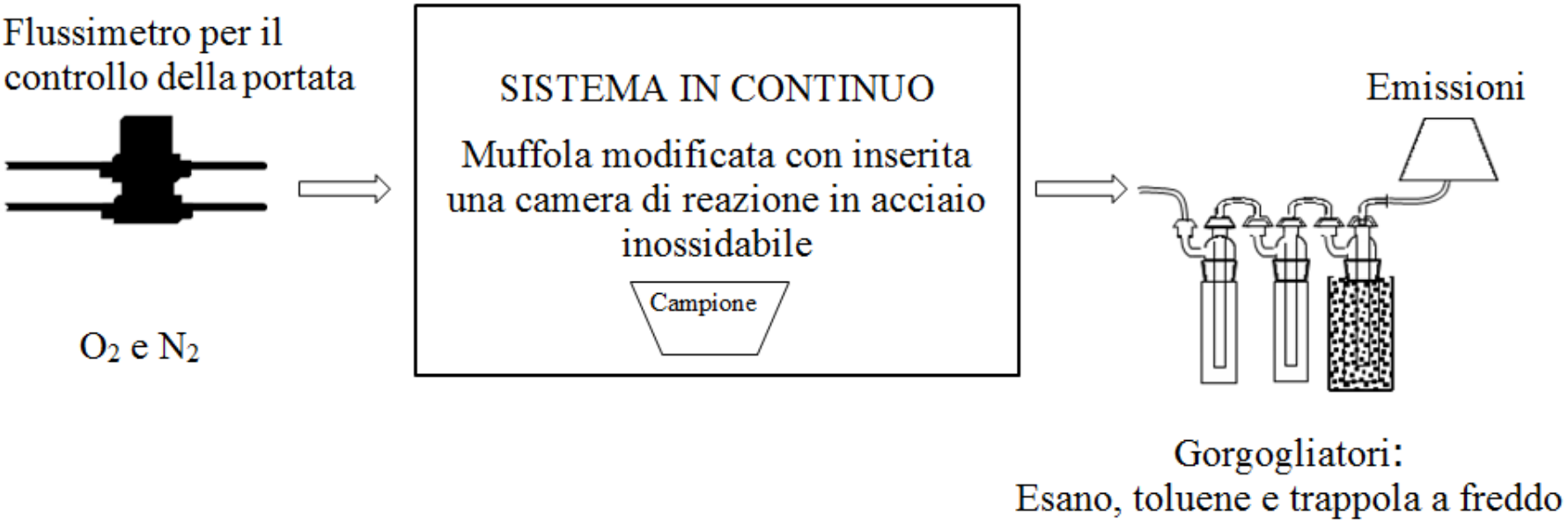
caratterizzazione di fly ash

studio della reazione di gassificazione del carbonio nativo

- CO_2, CO
- *Total Organic Carbon*
- PCDD/F

Flow-over-solid-system

Simulare le zone fredde della linea fumi



Trattamento termico

- Muffola, modificata per l'inserimento della camera di reazione in acciaio inox;
- Flussimetro per il monitoraggio della portata d'aria in ingresso;
- Sistema di controllo della T di reazione (termocoppia).

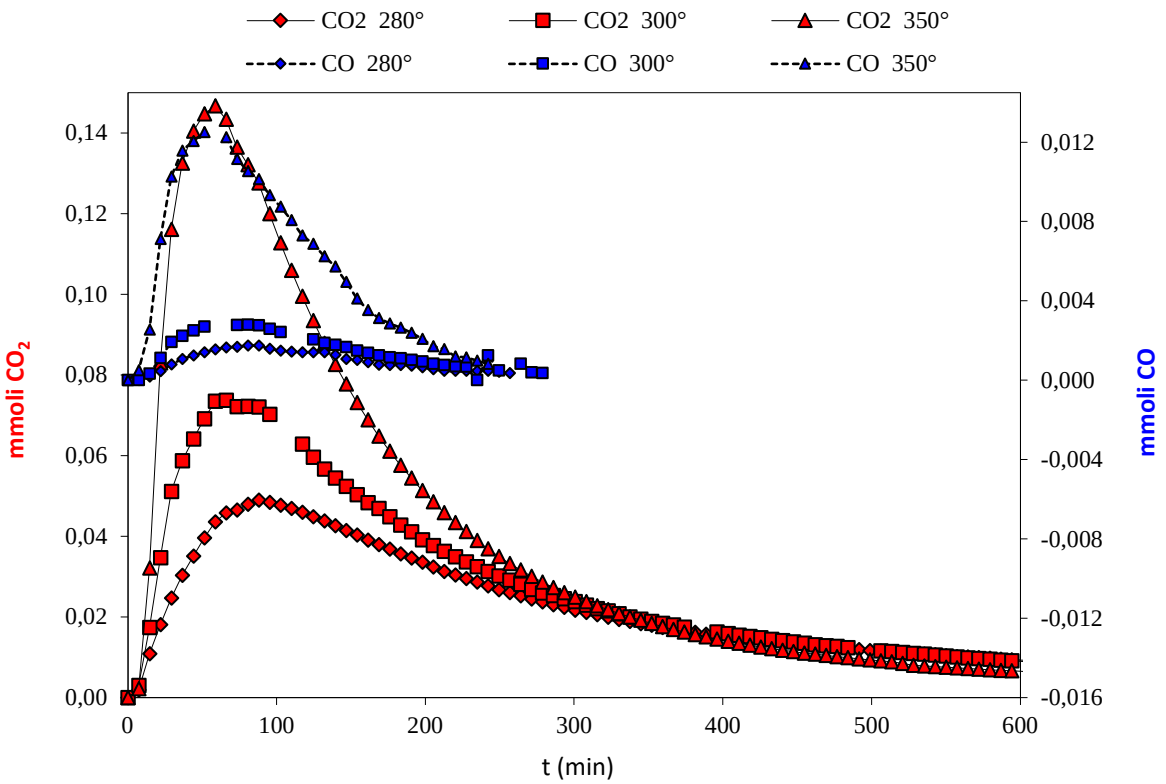
Condizioni operative:

Reagenti: FA (substrato) e aria sintetica (100 mL/min)

Temperatura di reazione: T = 280°C

Tempi di reazione: 9 h, 15 h, 21 h

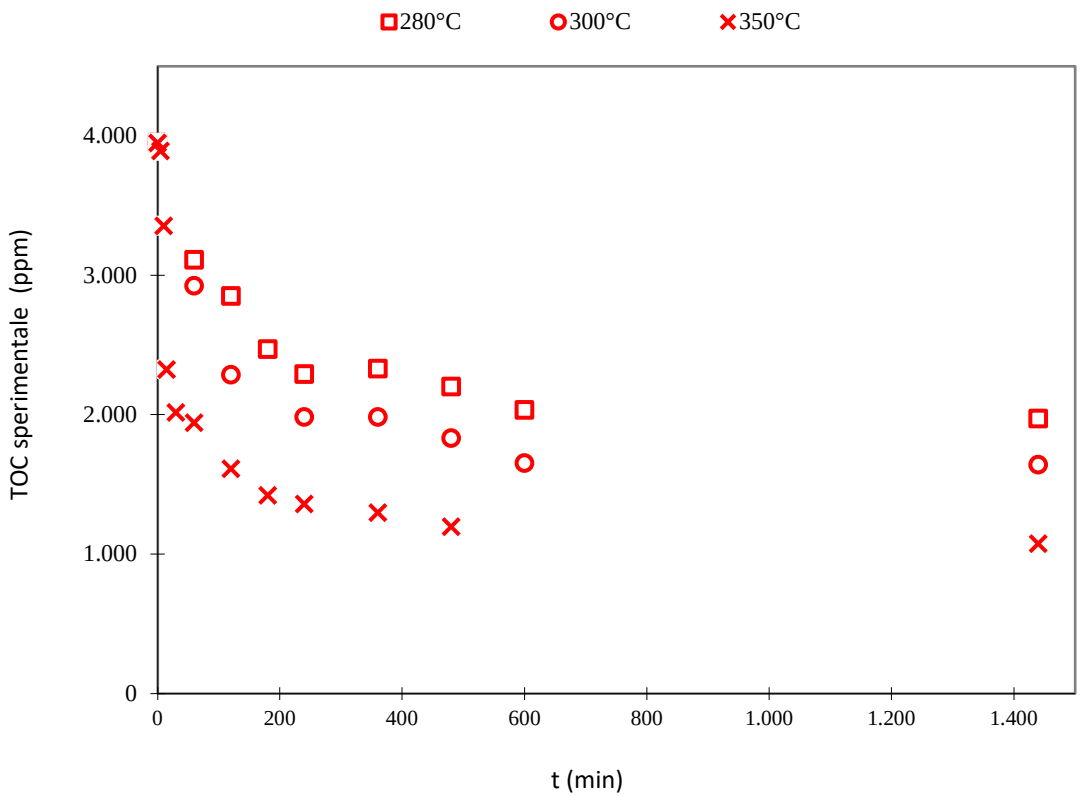
CO_2 e CO



- formazione CO fino a 4 ore
- CO_2 circa il 95% del totale
- max CO e CO_2 a tempi simili

l'ossidazione del carbone nativo è il risultato di due reazioni parallele; il ruolo di CO può essere ritenuto trascurabile

carbonio

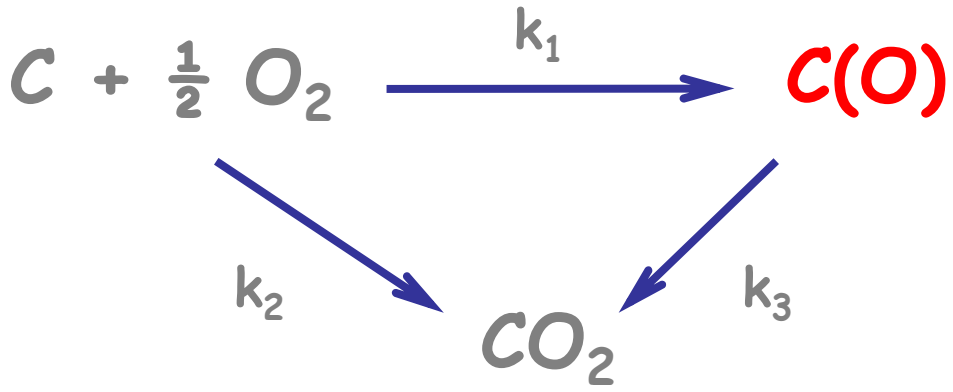


carbonio misurato come TOC
→ *total organic carbon*
«lump parameter»

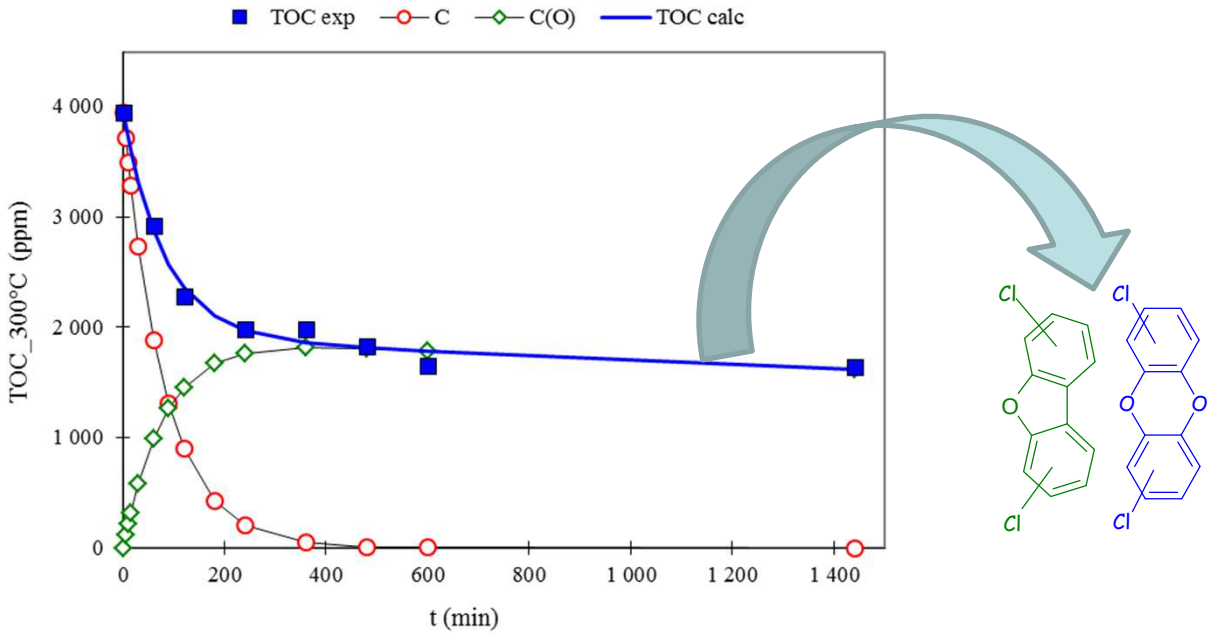
dopo 600 min:

- ❑ il TOC residuo rimane praticamente costante
- ❑ non si osserva più formazione di CO_2

Modello cinetico

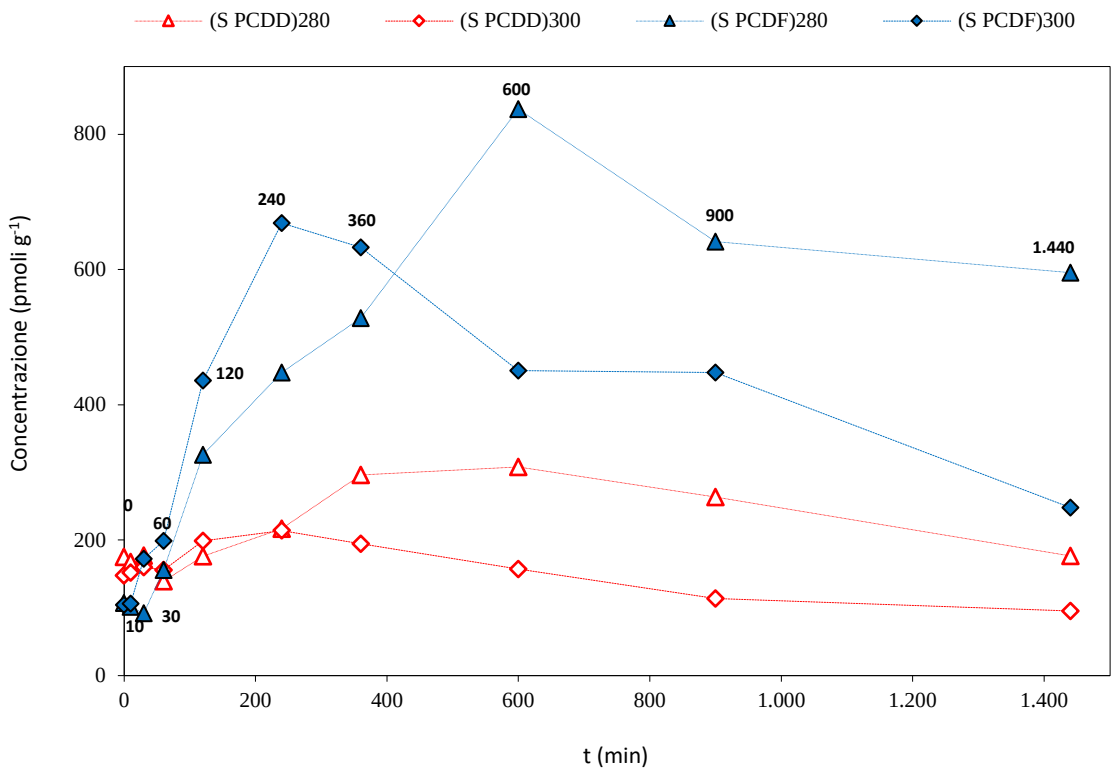


$$\frac{TOC}{TOC_0} = \frac{[C]+[C(O)]}{[C]_0} = \frac{k_1}{(k_1+k_2-k_3)}\exp(-k_3 t) + \frac{(k_2-k_3)}{(k_1+k_2-k_3)}\exp[-(k_1+k_2)t]$$



PCDD/F

in fase vapore:
concentrazione di PCDD/F
trascurabile



- ❑ max concentrazione a 600 min a 280°C e a 240 min a 300°C
- ❑ PCDF/PCDD > 1 dopo 30-60 min
  maggiore abbondanza % di congeneri con alto grado di clorurazione

→ sintesi de novo di PCDD/F
→ bilancio di formazione e distruzione

Modello cinetico

Modello cinetico sviluppato considerando costanti
le concentrazioni di ossigeno e cloro
(presenti in forte eccesso)

$C_{DF} \rightarrow (PCDD/F)_s$ (k_a)

$(PCDD/F)_s \rightarrow P$ (k_p)

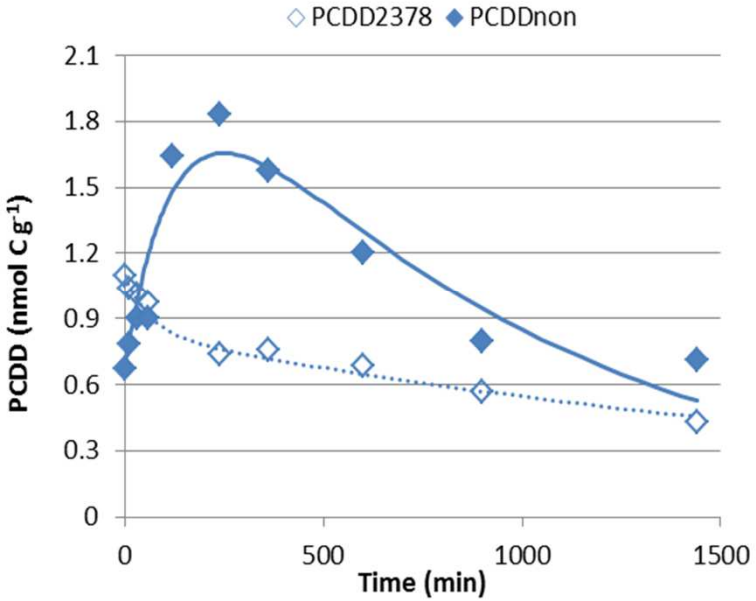
$$[(\mathrm{PCD\,D/F})\mathrm{s}] = [(PCD\,D/F)\mathrm{s}]_0 e^{(-k_p t)} + [C_{DF}]_0 \frac{k_a}{k_p - k_a}\left[e^{(-k_a t)} - e^{(-k_p t)}\right]$$

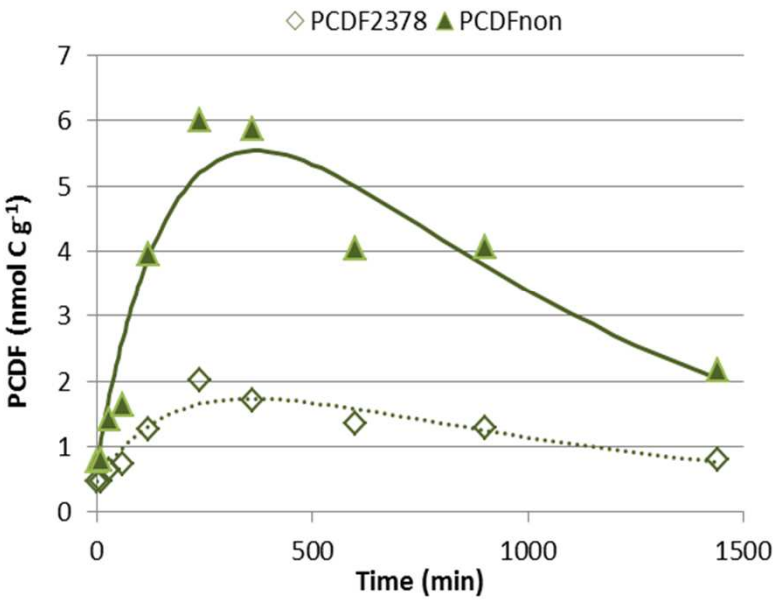
k_a costante cinetica delle reazioni di formazione

k_p costante cinetica delle reazioni di distruzione (ossidazione, declorurazione e decomposizione)

$C_{[DF]0}$ frazione di carbonio nativo coinvolta nella reattività di PCDD/F

Modello cinetico





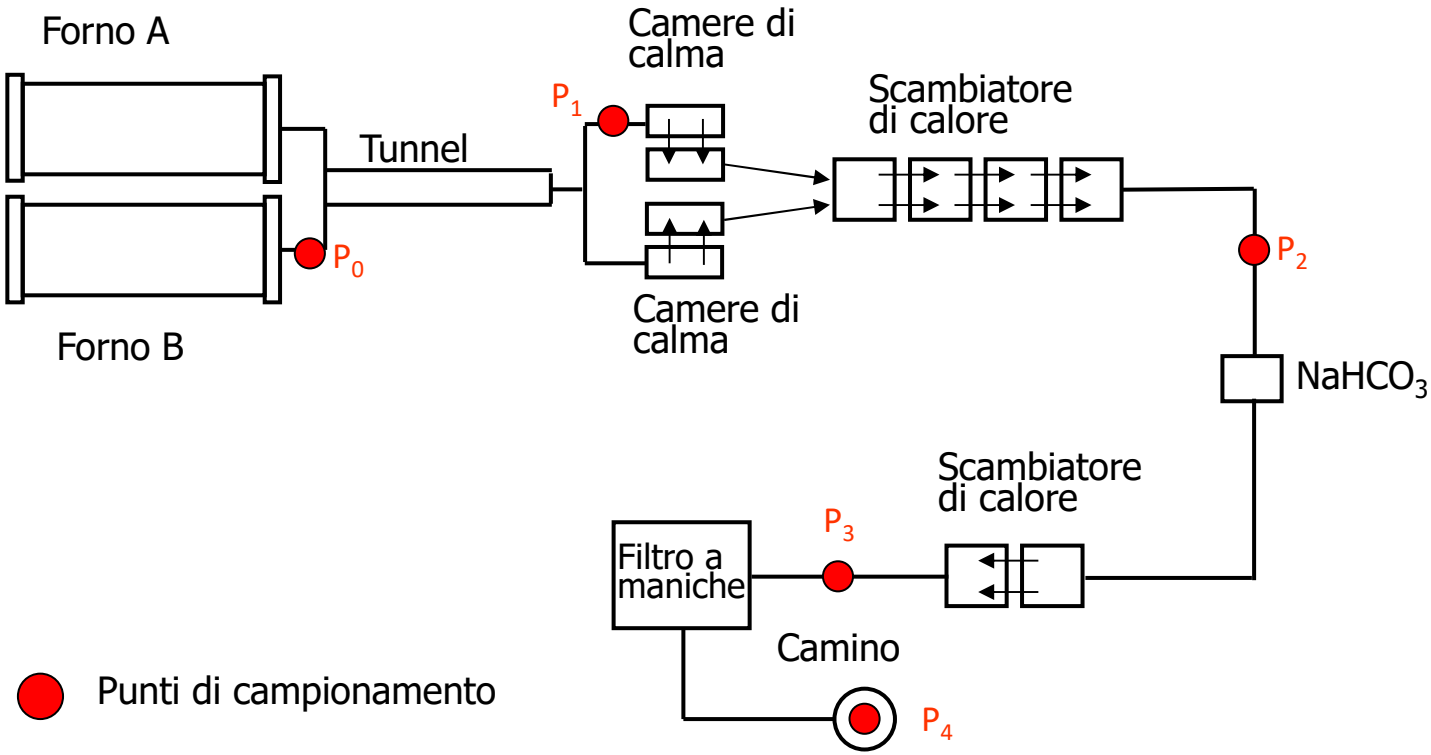
→ buon accordo tra le curve calcolate e i valori sperimentali per gli omologhi PCDD2378, PCDDnon, PCDF2378 e PCDFnon

→ i valori delle costanti cinetiche k_1 e k_a sono simili

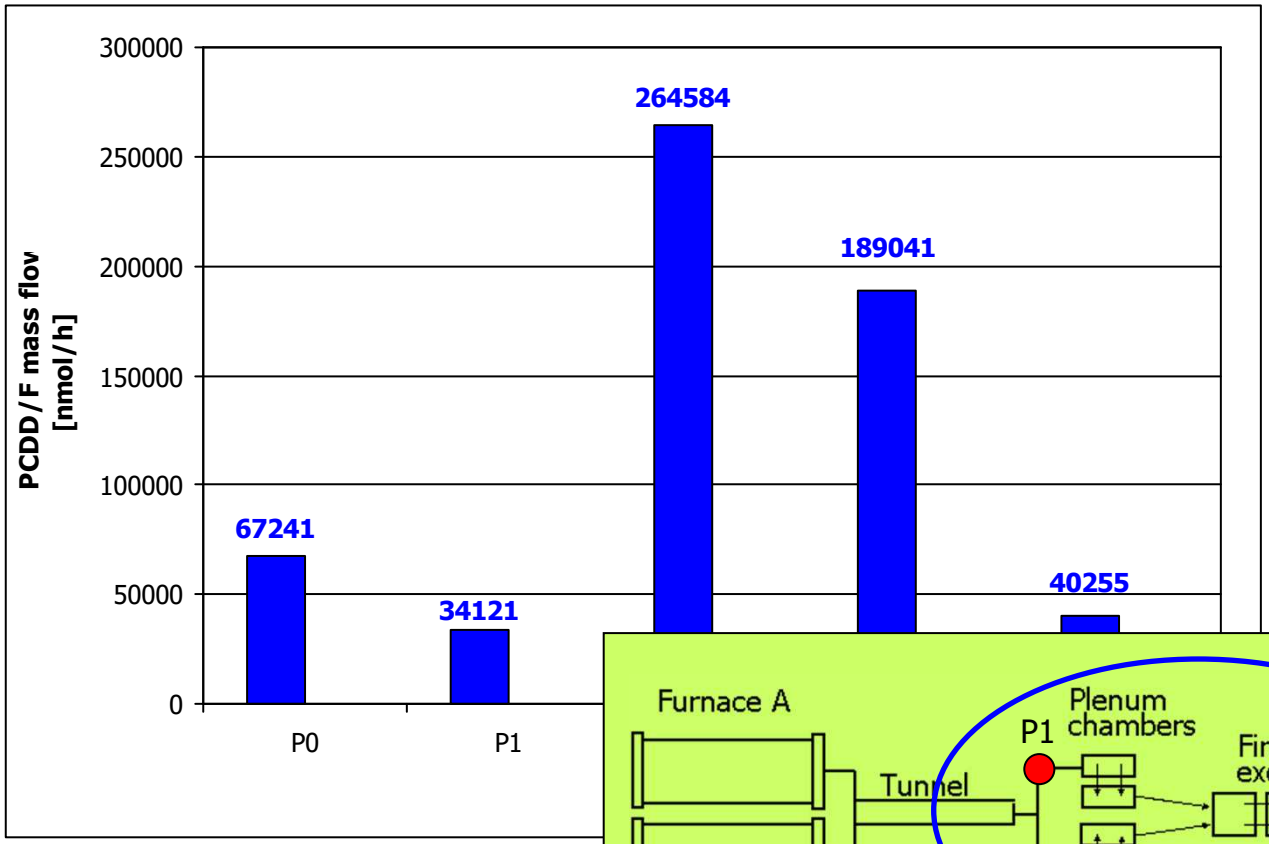
→ la reazione di ossidazione del carbone nativo e la formazione di PCDD/F hanno in comune la formazione dei complessi ossigenati C(O)

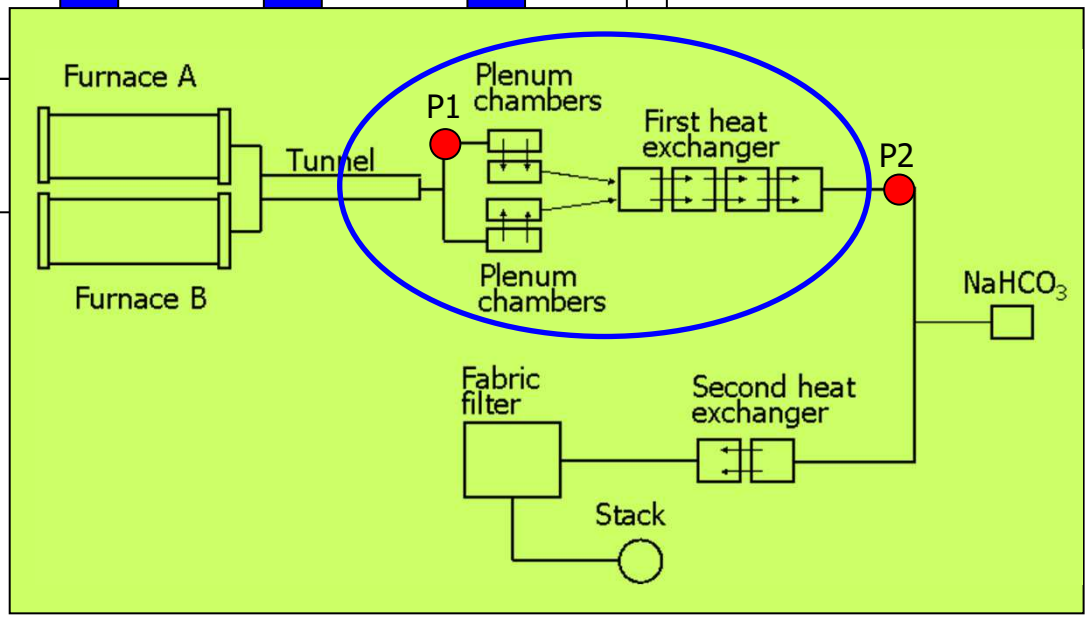
SCALA INDUSTRIALE

La sperimentazione effettuata su un impianto di seconda fusione dell'alluminio costituisce la ricaduta applicativa degli studi a scala di laboratorio

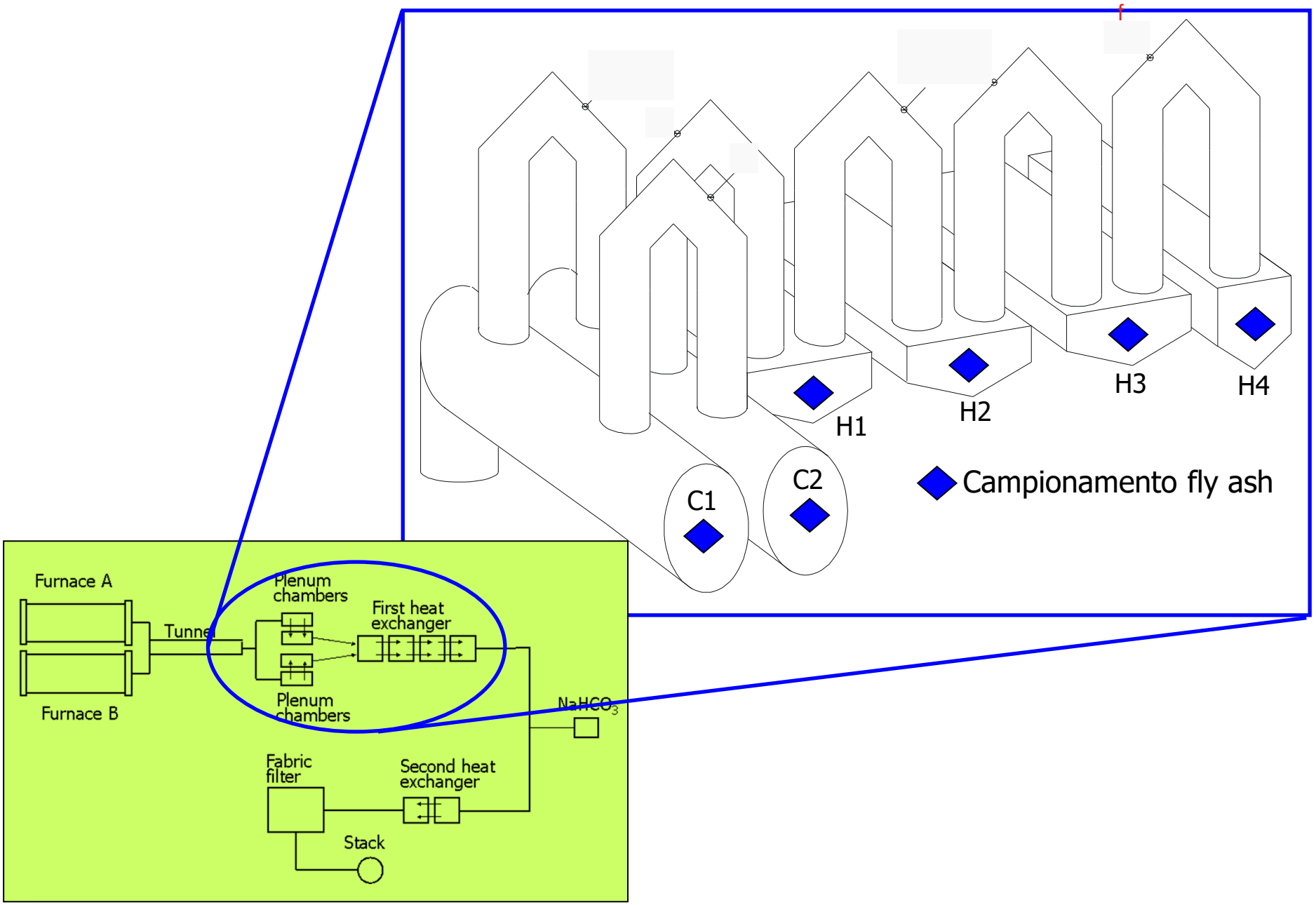


Flusso di massa [nmol/h] di PCDD/F lungo la linea fumi

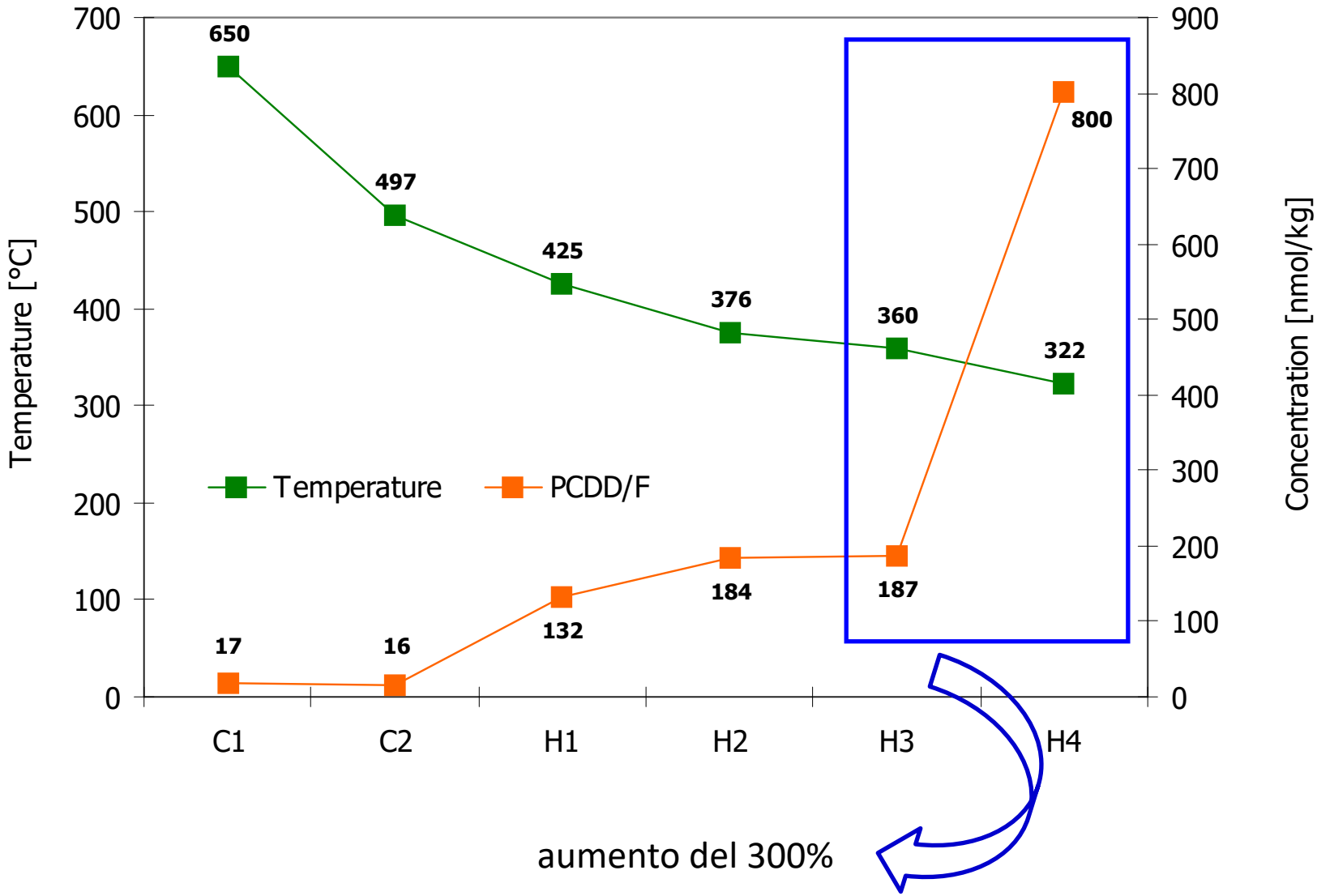




Focus su P1 – P2: camere di calma e tramogge del primo scambiatore

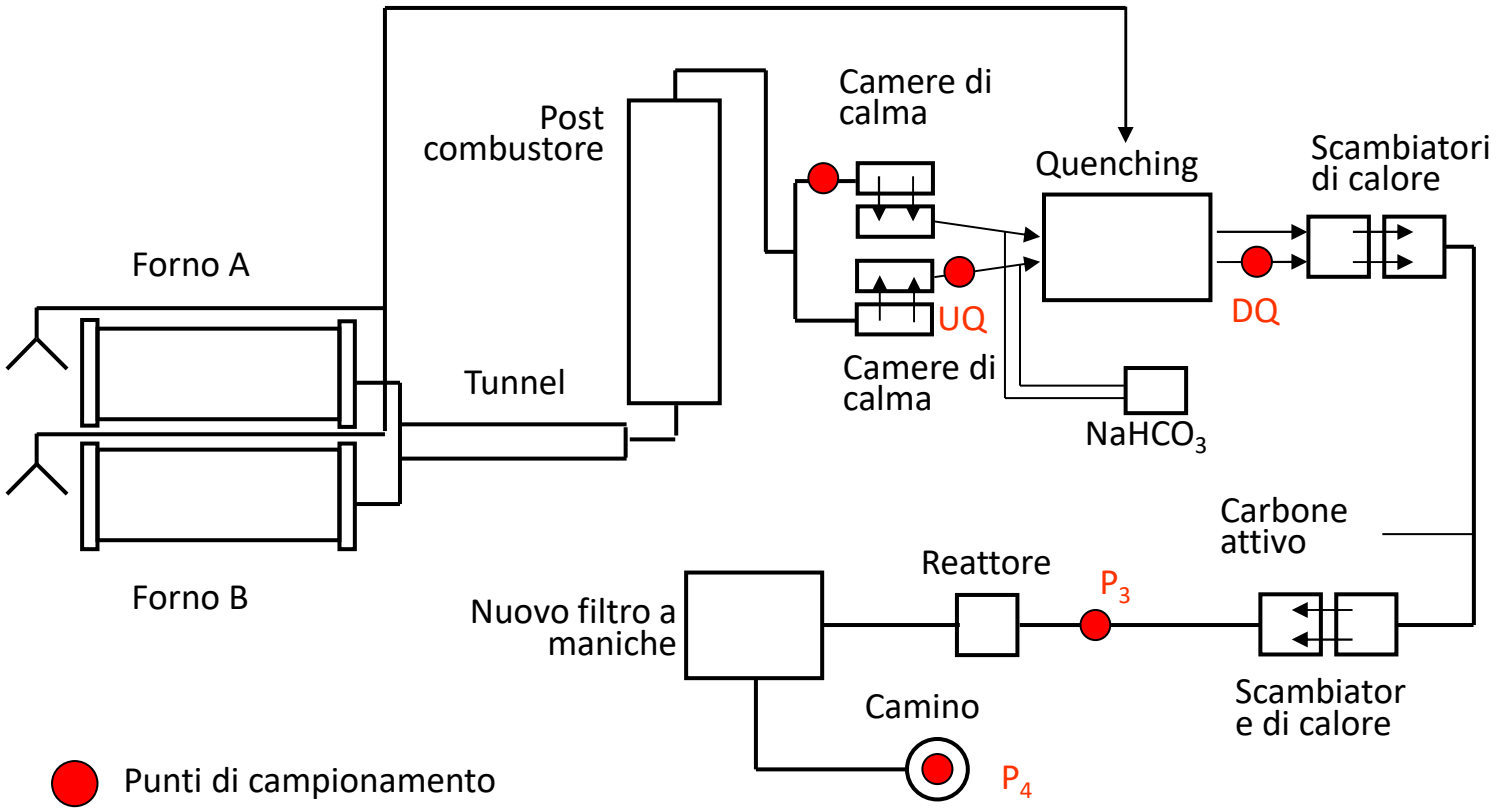


Correlazione tra PCDD/F e temperatura



Interventi sulla linea fumi

- ❑ installazione del post-combustore all'uscita del tunnel
- ❑ installazione del quenching tra P1 e P2
- ❑ sostituzione del filtro a maniche



Interventi sulla linea fumi

- installazione del **post-combustore** all'uscita del tunnel → riduzione di concentrazione di PCDD/F nei fumi in uscita pari a circa il 50%
- installazione del **quenching** tra P1 e P2 → minimizzazione del tempo di residenza dei fumi nell'intervallo 360 – 320 °C per prevenire la formazione di PCDD/F
- sostituzione del **filtro a maniche** → riduzione delle concentrazioni di particolato e PCDD/F all'emissione