

SUPREME COURT OF THE STATE OF NEW YORK
NEW YORK COUNTY

PRESENT: LOUIS B. YORK
J.S.C.
Justice

PART 2

Sean Keefe
-v-
BMA of North America

INDEX NO. 100725/08
MOTION DATE _____
MOTION SEQ. NO. _____

The following papers, numbered 1 to _____, were read on this motion to/for _____

Notice of Motion/Order to Show Cause — Affidavits — Exhibits _____ No(s). _____
Answering Affidavits — Exhibits _____ No(s). _____
Replying Affidavits _____ No(s). _____

Upon the foregoing papers, it is ordered that this motion is decided in accordance
with the accompanying decision.

MOTION/CASE IS RESPECTFULLY REFERRED TO JUSTICE
FOR THE FOLLOWING REASON(S):

FILED
MAY 15 2013
NEW YORK
COUNTY CLERK'S OFFICE

Dated: 3/10/13

Louis B. York
LOUIS B. YORK J.S.C.
J.S.C.

- 1. CHECK ONE: CASE DISPOSED NON-FINAL DISPOSITION
- 2. CHECK AS APPROPRIATE:MOTION IS: GRANTED DENIED GRANTED IN PART OTHER
- 3. CHECK IF APPROPRIATE: SETTLE ORDER SUBMIT ORDER
 DO NOT POST FIDUCIARY APPOINTMENT REFERENCE

SUPREME COURT OF THE STATE OF NEW YORK
COUNTY OF NEW YORK

-----x
SEAN REEPS, an Infant by His Mother and Natural
Guardian, DEBRA REEPS,

Plaintiffs,

Index No. 100725/08

-against-

BMW OF NORTH AMERICA, LLC, BMW OF
NORTH AMERICA, INC., BMW(US) HOLDING
CORP., MARTIN MOTOR SALES, INC.,
HASSEL MOTORS, INC.,

Defendants.

FILED

MAY 15 2013

-----x NEW YORK
COUNTY CLERK'S OFFICE

YORK, J.:

Plaintiffs move, pursuant to CPLR 2221(d) and (e) and 5701(c) for (a) a hearing (either at trial or pretrial) on the admissibility of plaintiffs' witnesses as to causation, Drs. Frazier, Bearer, Kramer, Adler and Sadler; (b) reargument or reconsideration of this court's decision of December 16, 2012 ("Decision") precluding the testimony of Drs. Frazier and Kramer; (c) renewal of the said decision, based upon new scientific evidence and new case authority; (d) an order granting plaintiffs the right to appeal the said decision to the Appellate Division, First Department.

Motion to reargue

Plaintiffs allege that this court misapprehended or ignored the factual record before it, impermissibly resolved credibility disputes between the parties' experts, and misapplied settled

*The Court expresses its thanks to Victoria Koroteyeva, Esq., for her excellent research and analysis of the scientific literature in connection with this motion.

legal precedent (George Aff. at ¶3). Counsel for plaintiffs lists 15 “facts overlooked by the court” (*id.*, at ¶¶ 27-31). The very first “fact” in this list misrepresents the court’s decision. The court allegedly stated that “plaintiff’s experts did not opine on quantification.” In reality, the court stated: “Plaintiffs’ experts expressed opinions on all three required elements of proof of causation.” (Decision, P.9). The reason counsel finds “mistakes” in the Decision is that she is not aware of differences between a “threshold” and a “dose-response relationship” (“mistake” No 6), between general and specific causation (“mistake” No. 7), does not know what a controlled epidemiological study is (“mistake” No. 8) or what “systematic” means (“mistake” No 14). In general, attorney for plaintiffs misrepresents the substance of this court’s Decision. The court did not prefer conclusions of defendants’ experts to that of plaintiffs – disagreement among experts is to be expected, since causation analysis involves professional judgment in interpreting data and literature. An expert opinion is precluded when it is reached in violation of generally accepted scientific principles. The court determined that Drs. Kramer and Frazier did not follow generally accepted scientific methodology.

Both experts did not cite a single scientific publication that establishes a causal link between exposure to gasoline vapors during pregnancy and the birth defects found in Sean Reeps. Gasoline is a common substance, frequently reviewed for toxicity by federal and state regulatory agencies. The Agency for Toxic Substances and Disease Registry (ATSDR), an agency of the U.S. Department of Health and Human Services, publishes detailed peer-reviewed analyses of potentially toxic agents based on all available scientific evidence. The volume on gasoline, which Dr. Frazier cites only for data about gasoline composition, did not find gasoline to be a developmental toxin (capable of producing defects in a developing fetus).¹ The State of California, under Proposition 65, The Safe Drinking Water and Toxic Enforcement Act of 1986,

¹ ATSDR. Toxicological Profile for Gasoline, PP. 37-38, available at <http://www.atsdr.cdc.gov/toxprofiles/tp72.pdf>

continuously updates its list of reproductive and developmental hazardous materials. The list is compiled by expert committees under peer review. The latest list, issued on April 19, 2013, does not include developmental effects of gasoline.² Contrary to established scientific practices, Drs. Kramer and Frazier pass over these negative results in silence. Instead, they claim to have found a causal link between gasoline and developmental outcomes that escaped other scientists.

Methods that Drs. Kramer and Frazier claim to use are those developed in epidemiology, including Bradford Hill criteria, as well as the general weight-of-evidence method and differential diagnostics. The court will cite the Reference Manual on Scientific Evidence, produced by the Federal Judicial Center and the National Research Council (hereafter “Reference Manual”) which sets forth accepted methodologies in epidemiology and toxicology.

Three basic issues arise when epidemiology is used in legal disputes, and the methodological soundness of a study and its implications for resolution of the question of causation must be assessed:

1. Do the results of an epidemiological study or studies reveal an association between an agent and disease?
2. Could this association have resulted from limitations of the study (bias, confounding, or sampling error), and if so, from which?
3. Based on the analysis of limitations in Item 2, above, and on other evidence, how plausible is a causal interpretation of the association?³

The authors of the reference guide on epidemiology offer a number of important caveats. These caveats address the same deficiencies in experts’ opinions pointed out in the Decision. It is well known that there can be no specific causation (the agent causing disease in a particular individual) in the absence of general causation (the agent is capable of causing disease).⁴ Epidemiology deals with general causation, specific causation is beyond its limits.⁵ The first

²State of California, EPA, Office of Environmental Health Hazard Assessment. Chemicals Known to the State to cause Cancer or Reproductive Toxicity, available at http://oehha.ca.gov/prop65/prop65_list/files/041913P65list.pdf

³ Federal Judicial Center, National Research Council. Reference Manual on Scientific Evidence, 3rd ed., 2011, Washington, DC: National Academies Press, P.554

⁴ *Id.*, P.552

⁵ *Id.*, P.553

question an epidemiologist addresses is whether an association exists between exposure to the agent and disease. The standard guidelines for inferring causation are based on Bradford Hill criteria. "These guidelines are employed only *after* a study finds an association to determine whether that association reflects a true causal relationship."⁶ (emphasis in the original). The Reference Manual criticized expert opinions in which experts "attempted to use these guidelines to support the existence of causation in the absence of any epidemiologic studies finding an association."⁷

Nowhere in their initial reports did Drs. Kramer and Frazier mention the fact that there are no epidemiological studies on the effect of *in utero* exposure to gasoline vapors and the kind of diseases found in Sean Reeps, or, more generally, birth defects. They gloss over this fact by referring to case reports which are not controlled epidemiological studies, and cannot establish association in the statistical sense used in epidemiology.⁸ Only in an affidavit in support of this motion did Dr. Frazier acknowledge the absence of epidemiological studies on the subject (Frazier Aff. of January 23, 2013, George Aff., Exh.B, at ¶32). It follows that no association between exposure to gasoline vapor in pregnancy and palsy, microcephaly, or congenital heart disease has ever been demonstrated, and thus the Bradford Hill causal criteria are not applicable (Decision, P.18). This central objection to experts' methodology in the Decision of the court is not addressed in Dr. Frazier's affidavit. There are scattered references to Bradford Hill criteria throughout the affidavit and even a special section illustrating how the expert applies them. What she calls a chart demonstrating application of Bradford Hill criteria to establish a causal link between gasoline vapors on the one hand, microcephaly and white brain matter injury on the other, does not deal with gasoline vapor at all, but with toluene (Frazier Aff. at ¶44). A closer look at the chart and works

⁶ *Id.*, PP.598-599

⁷ *Id.*, P.599, n.141

⁸ "An association between exposure to an agent and disease exists when they occur together more frequently than one would expect by chance." *Id.*, P.566.

referenced in it shows that she measures the strength of association between exposure to toluene and birth defects based on case reports, not epidemiological studies – a practice firmly rejected in the scientific community. In addition, the expert conflates general and specific causation, misinterpreting what “alternative explanations” for the Bradford Hill causal analysis mean. The Reference Manual, which she cites as her source, clarifies that these alternative explanations refer to problems of bias and confounding in an epidemiological study, relevant to general causation, and not alternative explanations for plaintiff’s injury, relevant to specific causation.⁹ Neither epidemiological methods in general, nor Bradford Hill criteria in particular, are methodologies actually used by Drs. Kramer and Frazier.

The two experts use a catch-all term the “weight of evidence” method (WOE) as a fall-back solution when they do not find supporting epidemiological evidence. The weight of evidence method is used in medical literature either in a rigorous scientific or metaphorical sense. It is used as methodology “where WOE points to established interpretative methodologies (e.g., systematic narrative review, meta-analysis, causal criteria, and/or quality criteria for toxicological studies) or where WOE means that ‘all’ rather than some subset of the evidence is examined, or rarely, where WOE points to methods using quantitative weights for evidence.”¹⁰ The metaphorical use of the term is, if nothing else, “a colorful way to say ‘the body of evidence we have examined and judged using a method we have not described but could be more or less inferred from a careful between-the-lines reading of our paper.’”¹¹

It is in the latter sense that Drs. Kramer and Frazier apply the term. The between-the-lines reading of their initial submissions led this court to methods actually used by plaintiff’s experts. Their first “method” is to substitute “gasoline vapors” in any statement of causation by “gasoline vapor and/or its volatile constituents.” Dr. Kramer follows this route: “There is ample epidemiological evidence to support that maternal exposure to gasoline vapor and/or its volatile

⁹ *Id.*, PP. 600 and 605.

¹⁰ Weed, D.L. Weight of Evidence: A Review of Concept and Methods, *Risk Analysis*, Vol. 25, No. 6, 2005: 1545.

¹¹ *Id.*, at 1546-1547.

constituents is capable of causing birth defects and other adverse birth outcomes among children exposed *in utero*.” (Kramer Aff. of December 9, 2010, at ¶11). Assuming that at least one element of gasoline could be shown to have teratogenic effect (causing damage to a developing fetus), the statement is not immediately false, though not necessarily true. Dr. Kramer does not mention gasoline vapors on their own. This is why this court found that she failed to state, let alone demonstrate, a causal link between exposure to gasoline vapors and birth defects (Decision, P.14). The other “method,” preferred by Dr. Frazier, is to shift among gasoline, organic solvents in general, selected organic solvents (BTEX), toluene and other elements indiscriminately, combining toxicological studies of rodents, case reports and scant epidemiological evidence (Decision, P.18). Both routes attribute to gasoline vapors the effects of toluene.

A series of case reports have shown that pregnant women who sniff toluene for its euphoric/hallucinogenic effects could have offspring with serious morphological and developmental defects. If a causal link between inhaling toluene and birth defects could be established, plaintiffs’ experts are confident that the same is true for gasoline because toluene is one of gasoline’s ingredients. As with gasoline, the two experts ignored the ATSDR assessment of toluene. After analyzing the same case reports as Drs. Kramer and Frazier, ATSDR experts concluded that:

The reports of birth defects in solvent abusers suggest that high-level exposure to toluene during pregnancy can be toxic to the developing fetus. The available human data, however, do not establish causality between low-level or occupational exposure to toluene and birth defects, because of the small sample size and the mixed solvent exposure experienced by the subjects in the Holmberg (1979) study, the lack of other studies of possible birth defects in children of occupationally exposed women, and the likelihood that the high exposure levels

experienced by pregnant solvent abusers (4,000–12,000 ppm) overwhelm maternal protection of the developing fetus from absorbed toluene. Experiments with pregnant mice demonstrated that 10-minute exposures to 2,000 ppm resulted in low uptake of toluene into fetal tissue and suggest that, at lower exposure levels, absorbed toluene is preferentially distributed to maternal adipose tissue before distribution to the developing fetus (Ghantous and Danielsson 1986).¹²

A responsible scientific inference from the evidence is a *suggestion*, not a conclusion, that toluene can have a toxic effect at high exposure levels that are also damaging to the nervous system of toluene abusers.

In their “weight of evidence” analysis Drs. Kramer and Frazier assume that toluene has been proven to be a developmental toxin, despite the lack of certainty about it in the scientific community. They proceed by arguing that case reports on toluene abusers are relevant to *in utero* exposure to gasoline vapors. Though the governmental report published in 1997 clearly stated that gasoline’s ingredients, such as toluene, ethylbenzene, xylene and benzene (BTEX) taken together, accounted for no more than 2% of gasoline vapor (cited in Kramer Report, at ¶23), they try to minimize this fact. Given that death may result after inhaling 5000 ppm of gasoline vapor,¹³ that toluene abusers inhaled 4000-12000 ppm of toluene, and toluene does not constitute more than 1% of gasoline vapor, the numbers simply do not add up. Dr. Frazier states that “the proper scientific methodology for evaluation of the toxicity of gasoline is to consider it fully as a mixture. It is not proper to conclude that a single agent needs to be present in a vapor at an extremely high concentration” (Frazier Aff. at ¶24) because of additive and interactive effects of other gasoline components. On her account, toluene in gasoline is more toxic than on its own due to the proven interaction between toluene and other elements of BTEX.

¹² ATSDR, Toxicological Profile for Toluene, PP.136-137, available at <http://www.atsdr.cdc.gov/toxprofiles/tp56.pdf>

¹³ ATSDR, Toxicological Profile for Gasoline, P.11

It is a standard practice in toxicology that common commercial mixtures, such as gasoline, are evaluated for toxicity a whole, not as a sum of its components.¹⁴ The ATSDR reports are an example of such an evaluation. Precisely because gasoline consists of more than 150 components, it is extremely difficult to take into account their combined effect. Dr. Frazier points only to the kinds of interaction between different components which strengthen the individual effects of each of them. In reality the study of mixtures identifies divergent outcomes:

When the effect of multiple agents is that which would be predicted by the sum of the effects of individual agents, it is called an additive effect; when it is greater than this sum, it is known as a synergistic effect; when one agent causes a decrease in the effect produced by another, the result is termed antagonism; and when an agent that by itself produces no effect leads to an enhancement of the effect of another agent, the response is termed potentiation.¹⁵

Dr. Frazier cannot assert with confidence that all components of gasoline produce synergistic, and not antagonistic effects or explain why inhalation of gasoline did not reveal the same effects as inhalation of toluene.

The key for establishing general causation is an estimate of a threshold exposure level which makes fetuses vulnerable to gasoline vapors. "For agents that produce effects other than through mutations, it is assumed that there is some level that is incapable of causing harm. If the level of exposure was below this no observable effect, or threshold, level, a relationship between the exposure and disease cannot be established."¹⁶ Neither Dr. Frazer, nor Dr. Kramer arrive at this threshold number (Decision, P.15). In her affidavit Dr. Frazier cites "threshold limit values" (TLVs) assessed by the American Conference of Governmental Industrial Hygienists. For whole unleaded gasoline it is 300 ppm (Frazier Aff. at ¶54). Dr. Frazier certainly knows what TLV means (a recommended air concentration below which no harm is expected for the average worker exposed at 8

¹⁴ Reference Manual, P.673

¹⁵ *Id.*

¹⁶ Reference Manual, PP.669-670

hours per day 5 days per week) (*id.*, at ¶25) but implies that this number is relevant to Mrs. Reeps' alleged exposure to gasoline vapors at 1000 ppm. The court was not misled by this implication. However plaintiffs' attorney misinterprets the meaning of TLVs: "Thus, the presence of symptoms of toxicity as described by Mrs. Reeps means that she was exposed to levels beyond the TLV and thus sufficient to be toxic to her, and thus her fetus" (George Aff. at ¶28). Both conclusions are not warranted. The Reference Manual on toxicology specifically warns: "Particularly problematic are generalizations made in personal injury litigation from regulatory positions. Regulatory standards are set for purposes far different than determining the preponderance of evidence in a toxic tort case."¹⁷ In this particular case the TLV did not even consider potential developmental effects of gasoline.

Plaintiffs' experts fail to establish a causal link between exposure to gasoline vapors above a threshold level and any birth defects using generally accepted scientific methods. This means that general causation was not proven. "[A]n agent cannot be considered to cause the illness of a specific person unless it is recognized as a cause of that disease in general."¹⁸ It would be contrary to sound scientific methodology to proceed to proof of specific causation without general causation. Drs. Kramer's and Frazier's attempts at "differential diagnosis," a method for establishing specific causation, are not relevant to this proceeding.

The process of differential diagnosis is undoubtedly important to the question of "specific causation". If other possible causes of an injury cannot be ruled out, or at least the probability of their contribution to causation minimized, then the "more likely than not" threshold for proving causation may not be met. But, it is also important to recognize that a fundamental assumption underlying this method is that the final, suspected "cause" remaining after this process of elimination must actually be capable of causing the injury. That is, the expert must "rule in" the suspected cause as well as "rule out" other possible causes. And, of course, expert opinion on this issue of "general causation" must be derived from a scientifically valid methodology.¹⁹

¹⁷ *Id.*, P. 665

¹⁸ *Id.*, P.613

¹⁹ Reference Manual, P. 613, citing Cavallo v. Star Enterprises, 892 F. Supp. 756, 771 (E.D. Va. 1995), *aff'd in relevant part*, 100 F.3d 1150 (4th Cir. 1996)

Dr. Frazier's estimate of Debra Reeps' exposure to gasoline vapors well illustrates her approach to the use of scientific methods and ways of citing literature. For proof of specific causation it is necessary to demonstrate that plaintiff's exposure to a harmful substance was above the threshold level capable of causing the disease. Dr. Frazier firmly states that this level in Debra Reeps' case was above 1000 ppm. The Decision explained why this assertion is problematic (Decision, P.19). Now Dr. Frazier insists that "generally accepted scientific methodology permits use of symptom thresholds to estimate exposure levels" (Frazier Aff., point C). Using the same flawed logic, she draws conclusions from studies showing how certain symptoms appeared at a certain exposure level, or increased with an increase in exposure. Reasoning on the model "If A, then B; B is present, so A is present as well" is a well-known logical fallacy. Unlike biological markers of exposure, individual reactions to an agent cannot serve to quantify exposure. "Acute exposure to many toxic agents produces a constellation of nonspecific symptoms, such as headaches, nausea, lightheadedness, and fatigue. These types of symptoms are part of human experience and can be triggered by a host of medical and psychological conditions. They are almost impossible to quantify or document beyond the patient's report."²⁰ Dr. Frazier is not discouraged by this conclusion. In her opinion, "In the clinical practice of occupational medicine, symptoms are used as a guide to judge exposure levels retrospectively because they generally correlate with indices of exposure." (id., at ¶37). The work that she cites as an example makes a directly opposite observation: "The odor recognition level for glutaraldehyde is 0.04 ppm. Eye and respiratory irritation are noted at different concentration levels depending on individual sensitivity."²¹

²⁰ *Id.*, P.671

²¹ Nayerbzadeh, A. The Effect of Work Practices on Personal Exposure to Glutaraldehyde among Health Care Workers, *Industrial Health* 2007, 45, 289-295, cited in Frazier Aff. at ¶37

On this motion to reargue the court does not find that it overlooked or misunderstood any of Dr. Kramer's or Dr. Frazier's statements. On the contrary, Dr. Frazier's affidavit confirmed that there are fundamental methodological problems with both experts' reports.

Neither did the court overlook or misapprehend the relevant New York State law on toxic torts and Frye hearing. Plaintiff's counsel proposes a reading of the case law that dispenses with several acknowledged principles. First, in counsel's opinion, there is no need to establish a threshold above which a substance may represent a health hazard (George Aff., P.11).

Toxicology's famous saying is "Dose makes poison"²² and the New York courts follow this principle. Coratti v Wella Corp., 56 AD3d 343; 867 N.Y.S.2d 421 [1st Dept 2008]; Fraser v 301-52 Townhouse Corp., 57 AD3d 416, 420; 870 N.Y.S.2d 266 [1st Dept 2008]; Cleghorne v City of New York, 99 AD3d 443, 447; 952 N.Y.S.2d 114 [1st Dept 2012] (plaintiffs' expert failed to posit the level of exposure necessary for the causation of injury). Counsel conflates different meanings of the term "dose," which the reference guide on epidemiology recommends keeping separate: "Evidence of a dose-response relationship as bearing on whether an inference of general causation is justified is analytically distinct from determining whether evidence of the dose to which a plaintiff was exposed is required in order to establish specific causation"²³. See, Decision, PP. 15-16 where the Court of Appeals' holding in Parker v. Mobil Oil Corp., 7 N.Y.3d 434, 824 N.Y.S.2d 584 [2006] was interpreted using this distinction. Next, counsel proposes a reading of Cornell v 360 W. 51st St. Realty, LLC, 95 AD3d 50, 60-61 [1st Dept 2012] which conflates general and specific causation. In the Cornell case, a number of agents, all known hazards, were connected with a number of diseases diagnosed in plaintiff. For the purposes of specific causation the court did not require a one-to-one relationship between a hazardous agent

²² Reference Manual, P. 636

²³ Id., P. p. 603, n.161

and a specific disease. In the present case it was not demonstrated that gasoline vapors are a developmental health hazard, so experts could not proceed to specific causation. The proposition for which the court cited Cornell (differential diagnosis is meaningless without general causation) is not marginal (Pl. Memo of Law, P.19) but goes to the heart of the relationship between general and specific causation.

Motion to renew

There are no new scientific facts or conclusions in the literature cited by Dr. Frazier that would lead the court to change its decision. Though Dr. Frazier framed her affidavit as a dialogue with defendants' experts, Drs. Scialli and Lees, in fact it is an attempt to salvage what remains of Drs. Kramer's and Frazier's argument after the court showed its flaws. The literature she cites (most of which was available at the time of the initial expert submissions) does not deal with the relationship between gasoline vapor and any of Sean Reeps' diseases and certainly does not state that gasoline is a developmental hazard.

There is no new case law that fundamentally change the way the New York courts' approach expert opinions on toxic torts. The Cornell case, which the court allegedly misunderstood, cannot serve as such new law. Cases from other departments which the court read prior to issuing its decision of December 16, 2012, are also not new, and deal with issues not relevant for this case. Thus the motion to renew is denied.

Request for an oral hearing on admissibility of plaintiffs' causation witnesses, Drs. Frazier, Bearer, Kramer, Adler and Sadler

As a preliminary matter, the court is not aware that a Frye hearing can be held to examine the party's experts at that party's request. The expert opinions of Drs. Bearer, Adler and Sadler

were not questioned by the opposite party, and these experts are not precluded from testifying at trial. On issues of general causation all of them deferred to Drs. Kramer and Frazier. The relevance of their testimony which, based on their pre-trial disclosure statements, concern issues of specific causation, is a separate matter.

As to Drs. Kramer and Frazier, the court had an opportunity to examine their opinions and cited literature based on two rounds of written submissions. With complex medical issues phrased in highly technical terms written presentations are a better way to get to the essence of the argument. The court has received a response to the issues it raised in its Decision from Dr. Frazier, and was confirmed in its criticism. A third attempt at extracting, from existing empirical research and published analysis, a statement on general causation relevant to the present case is not warranted. Though called a "hearing," a Frye hearing on written submissions is an accepted procedural device. Oppenheim v United Charities of New York, 266 AD2d 116; 698 N.Y.S.2d 144 (Mem) [1st Dept 1999]; Selig v Pfizer, Inc., 185 Misc 2d 600, 607; 713 N.Y.S.2d 898 [Sup Ct 2000] affd. 290 AD2d 319, 735 NYS2d 549 [1st Dept 2002](because the parties have totally exhausted the arguments and authorities in their submissions sufficiently in advance of the trial, the court could not see how a Frye hearing could shed any more light on the issues), see, also Ratner v McNeil-PPC, Inc., 91 AD3d 63, 67; 933 N.Y.S.2d 323 [2d Dept 2011].

Request for a leave to appeal

This court, exercising its discretion, refuses to grant leave to appeal its Decision of December 16, 2012. Plaintiffs have available to them an application to the Appellate Division for leave to appeal on the grounds listed in CPLR 5701.

CONCLUSION

For the foregoing reasons, it is

ORDERED that Plaintiffs' motion is denied in all respects

Dated: 5/10/13

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