EXHIBIT 28

FILED UNDER SEAL

IN THE CIRCUIT COURT
TWENTIETH JUDICIAL CIRCUIT
ST. CLAIR COUNTY, ILLINOIS

DIANA HOFFMANN, *et al.*, Plaintiffs, v. SYNGENTA CROP PROTECTION, LLC, *et al.*, Defendants.	No. 17-L-517

DECLARATION OF JON R. HEYLINGS, PHD

Under penalties as provided by law pursuant to Section 1-109 of the Illinois Code of Civil Procedure, I, Jon R. Heylings, Ph.D., certify based on my personal knowledge that the statements in this declaration are true and correct, except as to matters stated to be on information and belief, and as to such matters, I certify as aforesaid that I verily believe the same to be true.

1. Since May 2008, I have been the Chairman, Chief Scientific Officer, and co-owner of Dermal Technology Laboratory Ltd ("DTL"), an independent contract research organisation at Keele University in the United Kingdom ("UK"). At DTL, we use *in vitro* (non-animal) methods to study the absorption of chemicals following contact with the skin. We do this as part of testing the safety of new and existing chemicals, agrochemicals, personal care, and pharmaceutical products. We are also involved in research and development for new products aimed at improving the

delivery of drugs into and through the skin. In 2016, DTL received the Queen's Award for Enterprise in the category of international trade in recognition of our achievements in collaborating with major science-based companies around the world.

2. During the same period, I have also been an honorary Professor of Toxicology in the School of Pharmacy at Keele University, and worked with the Organisation for Economic Co-operation and Development (OECD), the World Health Organization, and several UK and European industry and government bodies to develop and advocate *in vitro* methods and test guidelines for evaluating the dermal absorption of chemicals.

3. From December 1986 through April 2008, I was employed at the Central Toxicology Laboratory (CTL),[1] a safety testing facility in the UK. When I joined CTL, it was owned and operated by Imperial Chemical Industries PLC ("ICI"), which later became Zeneca Group PLC ("Zeneca"), and eventually Syngenta AG ("Syngenta").

4. Between 1986 and 2008, I was employed by CTL in positions with progressively increasing research and managerial responsibility:

a. Research Scientist, Biochemical Toxicology Section (December 1986 to December 1992);

b. Work Group Leader, Investigative Toxicology Section, and CTL Quality Manager (January 1993 to June 1995);

[1] Previously known as ICI's Industrial Hygiene Research Laboratory.

c. Head of Absorption and In Vitro Toxicology, Metabolism and Pharmacokinetics Section (July 1995 to September 2006; appointed Senior Toxicologist, 1999); and

d. Head of Research and Investigative Toxicology Department (September 2006 to April 2008).

5. I left my employment with Syngenta when it closed CTL in 2008, but continued to undertake dermal absorption studies under contract for Syngenta for several years after leaving the company.

6. My career at CTL began in December 1986, when I was recruited from the University of Texas, Veterans Administration Medical Center in Dallas, where I had been a Postdoctoral Research Fellow in the Department of Gastroenterology since September 1985. Before I began my postdoctoral work in Texas, I had been employed as a graduate from September 1979 by ICI's Pharmaceuticals Division, where I earned my extramural Ph.D. in Gastroenterology in 1984 and was a Senior Experimental Officer in the Department of Bioscience Gastrointestinal Disease Unit.

7. My *curriculum vitae*[2] provides additional information about my education, appointments, membership and activities in professional associations, and over 100 external publications and 9 patents in the field of gastrointestinal and dermal science.

8. In 1986, I was recruited to join CTL by Dr. Lewis Smith, who was then the Head of the Biochemical Toxicology Section and the paraquat product manager for

[2] Exhibit 1.

CTL. My initial remit, and the purpose for which I was recruited, was to study the site and mechanism of paraquat absorption from the gastrointestinal tract. In that role, I designed, performed or supervised, and reported and published the results of *in vitro* studies investigating the site and mechanism of paraquat absorption from the gastrointestinal tract. The purpose of this research was to obtain knowledge about the absorption of paraquat in the gastrointestinal tract that might contribute to the development of safer formulations of paraquat—specifically, formulations less likely to kill a person who accidentally or intentionally ingests the product.

9. My other role, which I assumed during the first years of my employment, involved applying the knowledge gained from that basic research, along with the knowledge I had from my education and previous experience, to design, perform or supervise, and report the results of *in vivo* (animal) studies investigating potentially less toxic formulations of paraquat that were being screened for potential development by ICI's Agrochemicals Division. The identification of potentially "safer" paraquat formulations, including several of which I became patent holder, became my primary responsibility at CTL, and ultimately the focus of my career with ICI, Zeneca, and Syngenta over two decades.

10. From the outset, my responsibilities at CTL also included participating in and presenting the results of my research at various technical review meetings held at Jealott's Hill, the UK Technical Headquarters of ICI's Agrochemicals Division, as the

toxicology expert on the "Safer Paraquat Formulations" project team. In addition, I regularly presented my research data to other groups at ICI Agrochemicals at Fernhurst, the business headquarters of ICI Agrochemicals, who were charged with overseeing the stewardship of paraquat products.

11. Although the exact mix of these project teams varied depending on their purpose, they typically were comprised of both technical and business managers whose responsibilities included, among other things, product development, formulation research, and commercial and global product registration, and typically included high-level managers within "the business": ICI's Agrochemicals Division, which was accountable for paraquat stewardship as well as commercial aspects that included marketing, sales and profits. ICI Agrochemicals was the "client" and funding source for which CTL investigated the toxicology of and performed the regulatory studies on ICI pesticide products, including paraquat. The groups I was involved in from the early years of my tenure at CTL included, among others, the Paraquat Strategic Action Committee[3] Absorption of Paraquat – Safer Formulations Workgroup[4] and the Paraquat

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[3] Also known as the "PSAC." The PSAC was a high-level management group that was responsible for all aspects of worldwide strategy for ICI's paraquat business.

[4] The membership and work of the Absorption of Paraquat – Safer Formulations Workgroup is described in minutes of its meetings, e.g., Exhibit 2, SYNG-PQ-03714546 at 4665-4670; Exhibit 3, SYNG-PQ-03714546 at 4656-4664; Exhibit 4, SYNG-

Strategic Action Committee Toxicology Sub-Committee.[5] These groups worked on all aspects of paraquat registration and stewardship. On an annual basis, I also presented my results on new, potentially safer formulations of paraquat to the Technical Review Committee (TRC) at Jealott's Hill. The TRC, comprised of the Heads of each technical and commercial function within ICI Agrochemicals, was responsible for the direction of the research and for approving the development of new paraquat formulations, several of which were discovered at CTL, including Magnoxone and Inteon.

12. Despite previous efforts to reduce the number of fatal poisonings with paraquat products, which had included adding to Gramoxone a blue dye (to distinguish the product from beverages), stench (an offensive odor), and an emetic (a substance intended to induce vomiting) known as PP796, when I joined CTL in 1986, accidental and intentional paraquat poisonings were a serious problem for ICI Agrochemicals, as they had been in the previous decade. At the time, ICI was under great pressure from registration authorities and non-governmental organizations around the world to find a way to dramatically reduce the vast number of deaths

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PQ-03714546 at 4647-4655; Exhibit 5, SYNG-PQ-03714546 at 4639-4646; Exhibit 6, SYNG-PQ-03714546 at 4633-4638; Exhibit 7, SYNG-PQ-03714546 at 4626-4632.

[5] The membership and work of the PSAC Toxicology Sub-Committee is described in minutes of its meetings, e.g. Exhibit 8, SYNG-PQ-03714546 at 4566-4569; Exhibit 9, SYNG-PQ-03714546 at 4555-4558.

caused by the accidental and intentional ingestion of paraquat.[6] This pressure to address the paraquat poisoning problem continued to exist—and in some ways increased—during the more than two decades I worked at CTL.

13. As a result, throughout my years at CTL, those at ICI who were accountable for the safety of paraquat products, who made decisions affecting the safety of paraquat products, or both, including upper-level management at both CTL and the Agrochemicals business, understood—if not feared—that if paraquat continued to receive bad publicity due to poisonings round the world, the sale and use of paraquat products might be further restricted or even banned in certain territories, resulting in significantly decreased financial returns through reduced sales and/or increased costs.[7] This was not a speculative scenario; it had already happened by 1988,[8] and it happened again and again over the next 20 years, as one country after another tightened restrictions on the sale and use of paraquat products or banned them completely.

14. From 1987 through 1989, I designed, performed or oversaw, and reported the results of a large number of screening studies on potentially safer paraquat formulations. The vast majority of these studies used rats, which was the species used

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[6] See, e.g., Exhibit 10, SYNG-PQ-03714546 at 4671-4689; Exhibit 11, SYNG-PQ-02494164.

[7] See, e.g., Exhibit 3, SYNG-PQ-03714546 at 4656-4664, at 4657.

[8] Id.

to identify any new formulation type that was most likely to meet our objective for increased safety. The most promising formulations were then tested in dogs, which are a more relevant species for predicting the safety of paraquat formulations in humans, since they have a vomit reflex which rats do not have. During my time at CTL, a large number of new and potentially less toxic paraquat formulations were tested in dogs to determine if the blood levels of paraquat following a single oral dose were reduced compared with the same dose of the existing Gramoxone product that was on sale and responsible for many deaths round the world. I was personally involved in the design and interpretation of all these CTL dog studies on various formulations of paraquat through the 1990s and into the 2000s.

15. Our objective in these studies was to identify formulations that provided a clear safety factor of, ideally, at least 10X compared to ICI's flagship Gramoxone liquid concentrate,[9] a 200g paraquat ion per litre (20% weight/volume) solution with 500mg PP796 per litre (0.05% weight/volume, or 400:1 paraquat to PP796 weight/weight).

16. Because the toxicity of Gramoxone with 0.05% PP796 was our benchmark for evaluating the toxicity of potentially safer formulations, I became well-acquainted with the results of previous studies with this product (and of previous studies with

[9] A secondary objective was to identify formulations that provided at least a 5X safety factor. A candidate formulation's "safety factor" is the magnitude of the increased dose of paraquat that results in toxicity equivalent to the toxicity of the same dose of the benchmark formulation.

non-emeticized 20% paraquat concentrate), and acquired more knowledge about its toxicity from my own studies. I also became aware that data on the real-world effect of introducing the emeticized Gramoxone formulation, although limited, did not appear to support the safety factor ICI claimed the emetic should provide over the non-emeticized Gramoxone formulation. Simply put, I became concerned that the concentration of the PP796 emetic added to Gramoxone back in the 1970s was too low to be an effective emetic dose in man for the smallest volume of Gramoxone that could cause death if ingested: 10-15ml, or 2-3 teaspoons (the minimum lethal dose).

17. My scientific curiosity therefore led me to investigate how ICI had decided adding PP796 to Gramoxone at a concentration of 0.05% would provide an effective emetic dose of PP796 in humans who ingested the minimum lethal dose of Gramoxone. The historical paraquat reports I had seen did not describe in detail how this decision had been made. However, I knew the decision to use PP796 as an emetic in Gramoxone had its roots in the results of clinical trials on the compound, then known as ICI 63197, that had been done in the 1970s during its development as a drug by ICI Pharmaceuticals (like ICI Agrochemicals, ICI Pharmaceuticals was one of ICI's operating divisions), because the report on those clinical trials was among the

supporting references cited in the CTL report on the estimated effectiveness of PP796 in paraquat to cause vomiting in animals and man.[10]

18. Since I knew many of the Library and Information Group at ICI Pharmaceuticals, which was on the same campus as CTL, I requested and was granted access to the archives where ICI Pharmaceuticals stored its clinical trial data. I obtained a copy of the 1973 ICI Pharmaceuticals report[11] containing the human clinical trial data that had been used in 1976 to estimate that a 5mg dose of PP796 would cause vomiting in humans if ingested in 10ml of Gramoxone. I recognized immediately that the clinical trial data did not support that estimate. To the contrary, it was obvious data from the clinical trials had been "cherry-picked" to manufacture a dose-response relationship that could be used support the conclusion that the smallest dose of PP796 likely to induce vomiting, and thus prevent deaths caused by the ingestion of a minimum lethal dose of Gramoxone, was much lower—and thus less expensive—than the smallest dose of PP796 that could reasonably be inferred from the results of the clinical trials to be an effective emetic dose. No dose-response relationship could reasonably be inferred from the clinical trial data in human volunteers, the only trial that involved the systematic administration of a range of doses, in which only two of twelve volunteers vomited, one of them—the one who received the highest dose in the trial—well beyond the time

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[10] Exhibit 12, SYNG-PQ-00524793.

[11] Exhibit 13, SYNG-PQ-14420786_R.

required for vomiting to occur to prevent death from paraquat poisoning. Moreover, no minimum effective emetic dose or dose-response relationship could reasonably be inferred from the clinical trials data, even combining data from multiple trials. The drug was not being investigated for its emetic effect, so the trials were not designed to provide, either individually or collectively, the data required to reliably estimate the minimum lethal dose or the relationship between dose and emetic effect, and their design did not provide such data by chance.

19. In short, I discovered in 1990 that the dose-response relationship that ICI Agrochemicals had claimed for the emetic agent in humans for more than 13 years by then, and had used to establish the concentration of PP796 in Gramoxone that was expected to substantially reduce deaths caused by the ingestion of the product, was based on scientific misconduct, or more bluntly, a lie.

20. In 1990 and 1991, I repeatedly explained what I had found, and provided further support for my conclusions, both in meetings and in written memos to my manager, Dr. Smith (who later became the Director of CTL, and eventually Head of Development for Syngenta in Basel), as well as others, including upper-level managers, at both CTL and the Agrochemicals division. The explanations and supporting information in my memos from this period, which ICI and its successors have known about for almost—in many cases, more than—30 years, as well as the recipients' responses and my replies, speak for themselves, so rather than repeat myself and speak

for them, I have attached copies of these communications as exhibits to this declaration.[12]

21. I was not alone in urging as long ago as 1990 that the concentration of the emetic in Gramoxone be increased. On February 28, 1990, the ICI Agrochemicals Safer Paraquat Formulations Project team issued a progress report in which it made the following recommendation: "Consider the case for raising the level of emetic in current 'Gramoxone' formulations to improve safety margins."[13] The report acknowledged that "It has been found that increasing the concentration of the emetic in 'Gramoxone' by a factor of 5 resulted in a minimum of a 2-3 fold safety factor over standard 'Gramoxone.'"[14] It also noted the results from 5 years of monitoring poisoning cases after PP796 was added to paraquat formulations, observing that "There was no definitive evidence from this large database that inclusion of the emetic had resulted in a reduction in oral toxicity of paraquat."[15] The report admitted that "the original

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[12] Exhibit 14, SYNG-PQ-26134258 at 4258-4265; Exhibit 15, SYNG-PQ-26134258 at 4266; Exhibit 16, SYNG-PQ-26134258 at 4267-4268; Exhibit 17, SYNG-PQ-26134258 at 4270-4272; Exhibit 18, SYNG-PQ-26134258 at 4269; Exhibit 19, SYNG-PQ-26134258 at 4273-4274; Exhibit 20, SYNG-PQ-26134258 at 4275; Exhibit 21, SYNG-PQ-26134258 at 4276-4277; Exhibit 22, SYNG-PQ-26134258 at 4278; Exhibit 23, SYNG-PQ-03709681_R at 9698-9705.

[13] Exhibit 24, SYNG-PQ-02639780 at 9783.

[14] Ibid. at 9785.

[15] Ibid. at 9788-9789.

decision to add 0.05% emetic to GRAMOXONE was probably an underestimate of the effective emetic dose in man," observing that:

> The time-to-vomit parameter is extremely critical to remove non-absorbed paraquat. Recent studies suggest that animals must remove the herbicide within 20 minutes of ingestion in order to survive a lethal dose of paraquat. In order to achieve this, available data suggests that the minimum concentration of emetic in GRAMOXONE should be some 5 times higher than currently used."[16]

22. Notably, under the heading "Strategy," this February 1990 report discussed the pros and cons, from product safety and business perspectives, of a "proactive" approach—promoting a safer formulation in all markets—versus a reactive approach—keeping safer formulations "on the shelf" to provide a "fall-back option" if and when existing product registrations were threatened; as the report makes clear, ICI opted for the less-expensive reactive approach: to offer a safer formulation only if and when registration authorities made this the only for ICI to keep selling Gramoxone.[17]

23. At that time, ICI was well aware that increasing the concentration of PP796 in Gramoxone was feasible. As indicated in an October 26, 1990 memo from myself to Dr. Smith, in the formulation it had registered in France in the 1980s, ICI had increased the concentration of PP796 by threefold, and had reduced the concentration of paraquat in the product by half, to 100 grams of paraquat ion per litre. Together, these

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[16] Ibid. at 9799.

[17] Ibid. at 9811-9812.

two changes increased the ratio of emetic to paraquat by sixfold, and were found in toxicity studies to significantly decrease the toxicity of the French product relative to Gramoxone and to increase survival at what would be considered lethal doses by volume of standard Gramoxone.[18]

24. In a March 1, 1995 email to Andy Cook, Zeneca's Paraquat Product Manager, with a copy to Martin Wilks, its Product Medical Advisor, I again pointed out that the clinical trial data did not support the purported effectiveness of the emetic at a concentration of 0.05g/l in Gramoxone, that "a 3-5 fold increase in emetic concentration will markedly improve the efficiency of emesis in man," and that "by extrapolation this would suggest a 5 fold improvement in oral toxicity."[19]

25. Similarly, in a September 2000 internal email, I explained that "a concentration of 2.4mg/ml PP796 would cause vomiting within 30 min in a minimally lethal dose of Gramoxone" and that the "2.4mg/ml emetic version of Gramoxone provided a 5-fold safety factor in the dog (CTL/R/1250)," and could be expected to provide a similar safety factor in man.[20]

26. Despite their knowledge of the substantial risk of death to persons unlucky enough to ingest paraquat, the concentration of the emetic in Gramoxone was

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[18] Exhibit 25, SYNG-PQ-03709681_R at 9695-9697.

[19] Exhibit 26, SYNG-PQ-24557091 at 7093.

[20] Exhibit 27, SYNG-PQ-21802228.

never increased—not by ICI, not by Zeneca, and not by Syngenta—until Syngenta's 2008 introduction of Gramoxone Inteon (a formulation I am patent holder for), which included a threefold increase in the concentration of the emetic; a substance that forms a gel when it comes into contact with stomach acid (which increases the effectiveness of vomiting and slows the transit of paraquat to the jejunum, the primary site of absorption); and a purgative to speed the elimination of any paraquat that passes from the stomach into the small intestine. Gramoxone Inteon was shown to improve survival in human poisonings in a survey carried out by Syngenta in Sri Lanka,[21] and was registered in many countries around the world as a way of defending paraquat registrations that could be lost due to the large numbers of poisonings. It also had the potential to establish a new standard for reduced-hazard paraquat products (paraquat products are now formulated and sold by companies other than Syngenta).

27. Based on my experience as head of the research team working on safer formulations of paraquat at CTL for over 20 years, which included reading numerous internal documents addressing the subject, it is my considered opinion that the primary reason why ICI, Zeneca, and Syngenta waited until 2008 to launch a safer formulation of Gramoxone was that management within the Agrochemicals business (later renamed

[21] Exhibit 28, SYNG-PQ-00038660.

Crop Protection) were more concerned about the sales and profitability of their paraquat products than they were about human safety.

28. When I left CTL to form my own company in 2008, I was pleased that Syngenta had launched Gramoxone Inteon with its higher emetic level and that my teams' work over the previous decades would save lives. However, in 2018, when I became involved with paraquat once more in a 3-year UK Government project on skin decontamination, I checked the recent literature on paraquat as part of these new investigations. I learned to my surprise that Syngenta had withdrawn Gramoxone Inteon from the market. I also came across a California EPA publication, "One Sip Can Kill," which reported many accidental paraquat poisonings in the USA.[22] Most shockingly, I learned that the current United Nations Food and Agriculture Organisation (FAO), which publishes international standards for the composition of pesticide products in their Specifications, still specified that paraquat liquid concentrate products (like Gramoxone) contain PP796 "at not less than 0.23% of paraquat ion content."[23] According to the Specifications, this standard meets the requirement that "Emesis must occur in about half an hour in at least 50% of cases."[24] It does not; the

[22] Exhibit 29, HEYLINGS-0000000064_R.

[23] Exhibit 30, HEYLINGS-0000000020_R at 0029.

[24] Id.

0.23% floor set by the FAO Specifications is substantially the same as the original, 0.05% w/v concentration of PP796 that ICI and Chevron Chemical Company chose in 1976.[25] That concentration is a fraction of the concentration sufficient to cause emesis in about half an hour in at least 50% of cases, as ICI knew no later than 1990, and on information and belief, as ICI and Chevron Chemical Company knew as early as 1976.

29. When these facts came to my attention, I contacted Syngenta and asked them to explain how this state of affairs had come to be. After multiple teleconference calls and meetings with high-level management at Jealott's Hill and Basel, Switzerland (Syngenta's worldwide headquarters), in which I explained once again that the 0.05% concentration of PP796 in Gramoxone was too low to be effective in the minimum lethal volume of Gramoxone and why that was the case, Syngenta, despite the evidence I had provided to them, dismissed my concerns as unfounded.

30. In May 2019, in a written response to me, Syngenta stated that I was wrong, and said that my previous allegations had been thoroughly looked into at the time. They went on to claim that a published 1987 paper by Meredith & Vale[26] showed PP796 was effective as an emetic in Gramoxone at the 0.05% concentration or the weight equivalent of 2 grams of paraquat ion.

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[25] In fact, PP796 at 0.23% of paraquat ion content is equivalent to a concentration slightly *lower* than 0.05% w/v, which corresponds to 0.25% of paraquat ion content

[26] Exhibit 31, SYNG-PQ-00059882.

31. Syngenta is wrong. The source of the unpublished ICI data in the Meredith & Vale paper that Syngenta relies on to support its claim that 0.05% PP796 in Gramoxone will cause emesis within half an hour in at least 50% of cases was an ICI internal report, by Bramley and Hart in 1982, on an ICI survey of paraquat poisonings in the UK.[27] Importantly, the unpublished ICI data in the Tables presented in the Meredith & Vale paper came almost exclusively from poisoning cases involving Weedol, a granular paraquat product that even with no emetic is inherently safer than non-emeticised Gramoxone. When compared to Gramoxone, the Weedol product sold during the period this survey covered was very low-strength (2.5%, vs. 20% paraquat ion) and had a much higher—and thus much more effective—ratio of PP796 emetic to paraquat in its composition than Gramoxone, as well as being a solid rather than a liquid.

32. Syngenta also claims the Meredith & Vale paper was the basis for the statement in the current FAO Specifications that PP796 at 0.23% of paraquat ion content in paraquat liquid concentrates (like Gramoxone) will cause emesis within about half an hour in at least 50% of cases. Syngenta has not shown me any evidence that that FAO relied on the Meredith & Vale paper as the basis for this statement, and I am not aware of any. If it did, for the reasons just explained, that was a mistake.

[27] Exhibit 32, SYNG-PQ-03720006_R.

33. At the request of the attorneys for the Plaintiffs in this case, I have carefully reviewed nearly 200 100 ICI and Chevron Chemical Company documents regarding the addition of PP796 to Gramoxone and Chevron's paraquat concentrates that predate my time at CTL.[28] On information and belief, I assume these documents are what they appear to be. Given that assumption, the information in these documents not only confirms the conclusions I reached in 1990 about the dubious origin of 0.05% as the standard for PP796 in paraquat concentrates and management's base motive for repeatedly refusing to increase that standard to make these products less lethal if ingested, it provides further support for those conclusions.

Certified as set forth above this 14th day of March, 2021.

Jon R. Heylings, Ph.D.

[28] See Exhibit 33.

CURRICULUM VITAE

NAME Professor Jon R Heylings

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EDUCATION

1967-1974 Richmond Grammar School, North Yorkshire, UK
1975-1979 BSc (Hons) Medical Sciences, University of Bradford, UK
1980-1984 PhD Gastroenterology, ICI Pharmaceuticals Division, UK
1985-1986 Postdoctoral Research Fellow, University of Texas, USA

OTHER ESTEEM INDICATORS & APPOINTMENTS

1993 Johns Hopkins Center for Animal Alternatives Award (CAAT)
1995 Appointed Quality Manager for AstraZeneca CTL, Alderley Park
1999 Appointed Senior Toxicologist at AstraZeneca CTL, Alderley Park
1999 Appointed UK Representative for OECD Test Guideline Development
2002 Appointed as the Industry Member of the OECD Writing Group
2006 Appointed UK Representative for IPCS EHC 235, Dermal Absorption
2007 Chairman of Dermal Technology Laboratory Ltd, Keele University
2008 Appointed Honorary Professor of Toxicology, University of Keele
2008-present Advanced Toxicology Course Tutor, University of Surrey
2009-present MSc Course Lecturer, School of Pharmacy, Keele University
2011 External Examiner, Postgraduate Studies, University of Manchester
2012 Doctoral External Examiner, University of Birmingham
2014 External Examiner, University of Queensland, Australia
2014 PhD External Examiner, University of Hertfordshire
2019 Doctoral External Examiner, University of Dublin, Ireland

MEMBERSHIP OF LEARNED BODIES & PROFESSIONAL ASSOCIATIONS

1986-present Member of the British Toxicology Society
1992-present Member of the PPP Scientific Advisory Board
1995-1996 ECVAM Workshop Chair for Percutaneous Absorption
1995-2000 ECPA Toxicology Sub-Group for Risk Assessment
1996-2002 OECD Technical Committee for Dermal Absorption
1998-2002 ECVAM Dermal Toxicity Steering Committee
1999-2005 Biomedical Sciences Advisory Board, University of Bradford
2002-2003 EDETOX EU Dermal Absorption Project Reviewer
2010- Health Protection Agency, Dermal Absorption Review Committee
2016- ECPA and EFSA Advisory Groups on Dermal Absorption
2018- UK Representative, OECD Expert Group for Dermal Absorption

EMPLOYMENT RECORD

Dates	Location	Position
May 2008-Present	Dermal Technology Laboratory Ltd, Keele University Science and Business Park, Keele	Chairman and Chief Scientific Officer Honorary Professor of Toxicology, School of Pharmacy, Keele University
Sept 2006-April 2008	Syngenta CTL, Alderley Park, Macclesfield, Cheshire	Head of Department, Research and Investigative Toxicology
July 1995-Sept 2006	Zeneca /AstraZeneca, CTL, Alderley Park Macclesfield, Cheshire	Head, Absorption and In Vitro Toxicology, Metabolism and Pharmacokinetics Section (Senior Toxicologist Appointment 1999)
Jan 1993-June 1995	Zeneca CTL, Alderley Park, Macclesfield, Cheshire	Work Group Leader, Investigative Toxicology Section and CTL Quality Manager
Dec 1986-Dec 1992	ICI CTL, Alderley Park, Macclesfield, Cheshire	Research Scientist, Biochemical Toxicology Section
Sept 1985-Dec 1986	University of Texas, Veterans Administration Medical Center, Dallas, Texas, USA	Postdoctoral Research Fellow, Department of Gastroenterology
Sept 1979-Sept 1985	ICI Pharmaceuticals, Alderley Park, Macclesfield, Cheshire	Senior Experimental Officer, Gastrointestinal Disease Unit, Department of Bioscience

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2. Garner A and HEYLINGS J R (1979). Stimulation of alkaline secretion in amphibian-isolated gastric mucosa by 16, 16-dimethyl PGE_2 and $PGF_{2\alpha}$: a proposed explanation for some of the cytoprotective actions of prostaglandins. Gastroenterology **76** 497-503.
3. Flemstrom G, Garner A, Nylander O, Hurst B C and HEYLINGS J R (1981). Intraluminal acid prostaglandin E2 and glucagon stimulate duodenal epithelial HCO_3- transport in the cat and guinea pig. Acta Physiol Scand **112** 20A.
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5. HEYLINGS J R, Garner A and Flemstrom G (1981). Regulation of gastroduodenal HCO_3 - transport by luminal acid in amphibian isolated mucosae. J Physiol Lond **316** 58P-59P.
6. Rees W D W, Garner A, HEYLINGS J R and Flemstrom G (1981). Effect of carbenoxolone on alkaline secretion by isolated amphibian gastric and duodenal mucosa. Eur J Clin Inv **11** 481-486.
7. Flemstrom G, Garner A and HEYLINGS J R (1982). Stimulation of epithelial HCO_3- transport by low luminal pH in amphibian gastric and proximal duodenal mucosae *in vitro*. J Physiol Lond **329** 67P.
8. Flemstrom G, Garner A, Nylander O, Hurst B C and HEYLINGS J R (1982). Surface epithelial HCO_3- transport by mammalian duodenum *in vivo*. Am J Physiol **243** G348-G358.
9. Flemstrom G, Garner A, Nylander O, Hurst B C and HEYLINGS J R (1982). Control of duodenal epithelial HCO_3- transport *in vivo*. In: Electrolyte and Water Transport across Gastrointestinal Epithelia, edited by Case R M, Garner A, Turnberg L A and Young J A, New York: Raven, pp. 267-275.
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