Supreme Court No. 100390-1

No. 80662-9

COURT OF APPEALS
OF THE STATE OF WASHINGTON,
DIVISION ONE

ASHA SINGH, as personal representative of the estate of NARENDRA P. SINGH,

Petitioner,

v.

STATE OF WASHINGTON, a governmental entity; UNIVERSITY OF WASHINGTON, a Washington State entity; and JOHN DOES 1-5,

Respondents.

ON APPEAL FROM KING COUNTY SUPERIOR COURT

PETITION FOR REVIEW

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I. IDENTITY OF PETITIONER

Asha Singh, as the personal representative of the estate of her late husband, Dr. Narendra Singh, asks this Court to accept review of the parts of the Court of Appeals' decision designated below; reverse the Court of Appeals; and remand for a trial. Asha will refer to herself as *Singh* and to her late husband as *Dr. Singh*.

II. COURT OF APPEALS DECISION

The Court of Appeals issued its decision terminating review on August 16, 2021, and denied reconsideration on October 20, 2021. A copy of the decision is attached as Appendix A. A copy of the order denying reconsideration is attached as Appendix B.

Singh seeks review of the Court of Appeals' decision declining to reinstate her claims for breach of contract, tortious interference, and failure to pay teaching wages, all of which were dismissed on summary judgment.

III. ISSUES PRESENTED FOR REVIEW

This petition involves four issues of substantial public interest that this Court should determine under RAP 13.4(b)(4).

1. Creation of a unilateral contract governing intellectual property created by faculty employed by a public-research institution. For nearly a century, employees' rights in their inventions have sprung from the employment contract. In many employment contexts, an employee who develops intellectual property does not have a formal employment contract.

This Court reaffirmed in *Storti v. University of Washington*, 181 Wn.2d 28, 330 P.3d 159 (2014), the longstanding rule that employment policies may form a unilateral contract between a university and its faculty under traditional contract principles. For most, if not all, faculty employed by the University of Washington, any intellectual property developed by their research is principally governed by a single, self-entitled policy document. That document grants rights and protectable interests to faculty to manage, to control, to preserve, and to destroy their research data. But according to the University and the Court of Appeals, because that policy document does not create contractual rights, the University has unfettered discretion to do as it pleases with a faculty's research data and intellectual property—including, as it did here, intentionally destroying a core faculty member's novel cell line that promised to treat cancer and to further scientific research.

Should this Court grant review, reverse the Court of Appeals, and hold that—consistent with its prior decisions like *Storti* and its progeny—provisions in an employment-policy document may form a unilateral contract between the University and its faculty?

2. The University's intentional destruction of a core faculty member's cancer research breached the unilateral contract governing the faculty's rights to intellectual property. Public-research institutions exist to preserve, to increase, and to transmit knowledge for the general benefit. This is precisely why most major scientific breakthroughs in cancer and vaccine research occur at these institutions.

The University's employment policies granted core research faculty like Dr. Singh the rights to maintain, to manage, to control, and to preserve their research data and intellectual property. Those policies also granted faculty the ultimate right to destroy such data and property.

While employed by the University, Dr. Singh developed a novel cell line valuable for cancer research. He published scholarship on this cell line for the benefit of the scientific community. Yet the University unilaterally destroyed this cell line without Dr. Singh's knowledge or approval.

Should this Court grant review to determine this issue of substantial public interest whether the University may destroy a core faculty member's research data in violation of the unilateral contract granting the faculty rights to manage, to control, to preserve, and ultimately to destroy their own research data?

3. The University's termination of a licensing agreement involving Dr. Singh's research data tortiously interfered with his valid business expectancy of royalties. Research-faculty compensation is supported principally by grant funding and licensing revenue generated by the faculty's research data and intellectual property.

Dr. Singh agreed to assist the University to license the cell line he developed to third parties in exchange for a portion of the licensing revenue. The University licensed that cell line to a third party but refused to perform that agreement and later unilaterally

terminated it, despite Dr. Singh's financial interest in the agreement.

Should this Court grant review to determine whether the University may unilaterally terminate a contract in which one of its core faculty members holds a financial interest?

4. A faculty member's right to be paid wages for compensable teaching activities and classroom instruction. Persons employed in Washington have the right to be paid wages for their work. Core research faculty principally conduct research in laboratories but may also teach classes, in which case they are entitled to be paid for any teaching activities.

Dr. Singh both conducted research and taught classes in the University's Bioengineering Department. The University failed to pay him all his teaching wages for work performed in 2015, shortly before he took an extended medical leave.

There are perhaps hundreds, if not thousands, of non-tenured faculty employed at the University of Washington who both conduct research and perform classroom instruction. These faculty are integral to furthering the University's academic mission and to leading the next generation of scholars.

Should this Court grant review to determine whether core research faculty at the University have the same rights to be paid wages for their teaching activities as do tenured faculty—another issue of substantial public interest?

IV. STATEMENT OF THE CASE

A. A trailblazer in the field of DNA analysis, Dr. Singh pioneered a technique over three decades ago to analyze DNA damage in cells.

After completing his graduate studies in India at its top medical school, Dr. Singh immigrated to the United States to

begin a post-doctoral fellowship on DNA damage. CP 1299. That fellowship allowed Dr. Singh to pursue his childhood dream of understanding the aging process and its causes.[1]

Five years later, while working at the National Institute of Health, Dr. Singh pioneered a technique called the comet assay to analyze DNA damage at the cellular level. CP 1300. That technique has since become the gold standard for human biomonitoring. CP 1300.

Dr. Singh's groundbreaking research, which has led to many scientific breakthroughs in other fields, sought to find answers to the aging process, including cancer. CP 1299–1302. His seminal paper on DNA damage has been cited over 12,000

[1] See Narendra P. Singh, *The comet assay: Reflections on its development, evolution and applications*, 767 MUTATION RESEARCH 23, 23–24 (2016) (attached as Appendix C).

times.[2] To put that into context, the Watson and Crick paper that first established DNA structure (*i.e.*, the double helix) has been cited about 16,000 times.[3] This alone speaks to the innovative and influential nature of Dr. Singh's work and its lasting impact on the scientific community.

B. Dr. Singh became a member of the core research faculty in the University of Washington's Bioengineering Department over two decades ago, where he conducted cutting-edge research, taught classes, and mentored students.

Dr. Singh joined the core research faculty of the University's Bioengineering Department in 1998. CP 725. He was one of about 50 core faculty in the Department. CP 724. Research faculty primarily conducted research but were also

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[2] *See* Narendra P. Singh et al., *A simple technique for quantitation of low levels of DNA damage in individual cells*, 175 EXPERIMENTAL CELL RESEARCH 184 (1988). The amicus memorandum of Dr. Robert H. Heflich, expected to be filed in support of this petition, provides additional context to Dr. Singh's research and achievements.

[3] *See* J.D. Watson & F.H.C. Crick, *A Structure for Deoxyribose Nucleic Acid*, 171 NATURE 737 (1953).

allowed to teach core bioengineering classes, which Dr. Singh regularly did during his career. CP 739, 1300.

C. Dr. Singh's intellectual property, including the cell lines he developed, were governed by several university policies that granted him contractual rights and protectable interests.

Intellectual property created by faculty at public-research institutions has burgeoned in the last forty years. *See* Daniel R. Cahoy, *Toward a Fair Social Use Framework for College and University Intellectual Property*, 41 J.C. & U.L. 485, 489 (2015). Research in the bioengineering and biotechnology spaces has particularly fueled the development of intellectual property. For instance, Dr. Singh focused his research on the intersection of DNA, cancer, and the aging process. CP 1299–1303. That research then transformed itself into research data, such as cell lines—the fruits of Dr. Singh's laboratory efforts. CP 902–08, 917.

To manage the development of this property, the University drafted several policies and memoranda to govern the

rights to and interests in such property. Three documents principally governed the rights in that property vis-à-vis the faculty and the University: the Patent, Invention, and Copyright (PIC) policy; Administrative Policy Statement (APS) 59.4; and Grants Information Memorandum (GIM) 37. CP 844, 850–63, 877–98, 902–08.

The PIC and APS 59.4 policies required the faculty to share with the University all revenue from licensed inventions. CP 844, 852, 883.

GIM 37 established the "rights in research data" generated by faculty employed at the University. CP 844, 902. These rights included ownership, stewardship, and preservation. CP 903–04. Although the University generally owned all research data, principal investigators chiefly controlled access to their research data, and research data could only be destroyed by the principal investigator. CP 903–05. Because Dr. Singh was University faculty; the cell lines he developed at the University were "research data"; and he was the "principal investigator" for

the cell lines, GIM 37 granted rights and protectable interests in research data to Dr. Singh. CP 903, 906.

D. Shortly before he was forced to take an extended medical leave, Dr. Singh developed a novel cell line that promised to treat cancer. The University licensed this cell line to a company for scientific and financial benefits.

During his employment at the University, Dr. Singh wrote papers and conducted research on numerous DNA-related topics, including age-related changes in DNA damage and repair, the role of environmental factors in DNA damage, and fetal origins of adult disease. CP 1301–02. He eventually developed a novel cell line (called the ARTN-103) that showed characteristics of cancer stem cells, including resistance to chemotherapy and radiation. CP 917, 1278, 1592. This scientific breakthrough promised to support "research on cancer, in particular studies on cancer therapies, early disease identification, [and] drug targeting." CP 917.

In January 2016, Dr. Singh collapsed, became debilitated, and required an extended medical leave. CP 1403–05. He, along

with his wife Asha, returned to India to be near family while he attempted to recover. CP 1405.

During his medical leave, Dr. Singh was contacted by a company wanting to license the ARTN cell line. CP 1281–82. The University, after months of negotiations, ultimately contracted with Applied Biological Materials (ABM) to license the ARTN cell line Dr. Singh had developed. CP 1592–97.

E. During Dr. Singh's medical leave, the University covertly began decommissioning his lab, despite requests from fellow faculty to help maintain Dr. Singh's laboratory and ongoing research. Later, after Dr. Singh's death, the University terminated the ABM licensing agreement and destroyed the licensed novel cell line that he had developed.

Knowing how much Dr. Singh's research meant to the scientific community, his family—most of whom are physicians themselves—contacted the University during Dr. Singh's medical leave to obtain assurances that the University would maintain and preserve Dr. Singh's laboratory, including his ongoing research and cell lines. CP 1461–62. One of Dr. Singh's colleagues in the Bioengineering Department,

Dr. Gerald Pollack, and others agreed to help maintain the laboratory and the ongoing research. CP 1737, 2315, 2326.

The University rebuffed efforts from Dr. Singh, his fellow faculty, and former lab assistants to maintain and to preserve Dr. Singh's laboratory and research data. CP 605–14, 693–94, 782–83, 1195–98, 1235, 1406, 1461–62, 1737. It shut out Dr. Singh from maintaining the integrity of his own laboratory and research data while he was on medical leave. *Id.* And it secretly began decommissioning his laboratory. CP 1406, 1461–62.

Dr. Singh passed away in India in late 2016. Apparently because of Dr. Singh's death, the University refused to perform the ABM contract, unilaterally terminated it, and destroyed all of Dr. Singh's research data, including the ARTN cell line. CP 1583.

F. Singh sued the University. Despite the University's destruction of Dr. Singh's cell lines, its failure to perform the licensing agreement, and its failure to pay Dr. Singh all his wages, the trial court dismissed all of Singh's claims on summary judgment. The Court of Appeals affirmed the dismissal of all but one of those claims.

Singh, on behalf of her late husband's estate, sued the University for breach of contract, tortious interference, conversion, disability discrimination, and unpaid wages. Her first three claims were principally based on the University's destruction of Dr. Singh's ARTN cell line.

The trial court dismissed all of Singh's claims on summary judgment. CP 1641. The Court of Appeals, Division One, affirmed the dismissal of all but the disability-discrimination claim.

V. ARGUMENT WHY REVIEW SHOULD BE ACCEPTED

A. Review is warranted to correct the conflicts created by Division One's decision. Provisions in employment policies governing rights in research data generated at a public-research institution may form a unilateral contract between faculty and a university.

Contracts typically come in two forms: bilateral and unilateral. *Storti*, 181 Wn.2d at 35. A unilateral contract is formed when one party makes a promise, and the second party accepts that promise through performance of their end of the bargain. *Id.* at 36. Review is warranted because Division One's decision creates conflicts in the law regarding unilateral contracts in employment policies that give contractual rights to employees in their intellectual property. RAP 13.4(b)(1), (2).

This Court has long recognized that employment policies may form a unilateral contract between employers and employees. *See, e.g.*, *Thompson v. St. Regis Paper Co.*, 102 Wn.2d 219, 228–29, 685 P.2d 1081 (1984) (holding that provisions in an employee policy manual may create contractual

obligations); *Storti*, 181 Wn.2d at 36–38 (reaffirming *Thompson* and holding that University of Washington faculty established an enforceable contract based on the University's promise in an executive order to extend raises for meritorious performance).

These seminal cases have been cited hundreds of times by courts applying these fundamental principles of employment law in Washington. *See, e.g., Duncan v. Alaska USA Fed. Credit Union, Inc.*, 148 Wn. App. 52, 62, 199 P.3d 991 (2008); *Carlson v. Lake Chelan Cmty. Hosp.*, 116 Wn. App. 718, 733, 75 P.3d 533 (2003). These principles apply naturally in the context of a university employee's contractual right to benefit from inventions and novel research. *See United States v. Dublier Condenser Corp.*, 289 U.S. 178, 187, 53 S. Ct. 554, 77 L. Ed. 1114 (1933) ("The respective rights and obligations of employer and employee, touching an invention conceived by the latter, spring from the contract of employment.").

But most faculty, including Dr. Singh, do not have a formal employment contract with the University that governed

rights in the research data they generated. Research faculty cannot acquire tenure and all the concomitant tenure rights and protections. Like Dr. Singh, these faculty's employment relationship with the University instead consisted solely of various policy documents. So Division One's misapplication of the unilateral-contract law here, which conflicts with the cases cited above, warrants correction.

GIM 37 is a self-entitled policy document governing research data. CP 902–08. Dr. Singh was a faculty. The ARTN cell line he developed while employed by the University was "research data." CP 903, 906. And he was the "principal investigator" for that cell line. CP 903. GIM 37 thus applied to Dr. Singh and his research data.

GIM 37 was intended to establish the "rights in research data" generated by faculty at the University. CP 902. These rights included ownership, stewardship, and preservation. CP 903–04. The University generally owned all research data; the principal investigator generally controlled access to their

research data; and the research data could be destroyed only by the principal investigator. CP 903–05.

Contrary to Division One's decision, the University's labeling GIM 37 as a policy or guidance document does not automatically make it so. A unilateral contract may be created if the three traditional contract requisites of offer, acceptance, and consideration are satisfied. *Storti*, 181 Wn.2d at 36.

GIM 37 proclaimed that principal investigators like Dr. Singh "shall" determine who has access to research data; are "responsible" for the collection, management, and retention of research data; and are ultimately "responsible" for the destruction of research data. CP 903–05. This language shows the University's intent to be bound by its promise of granting these rights to the faculty. *Storti*, 181 Wn.2d at 36.

The University's offer in GIM 37 was accepted by Dr. Singh. Up until his medical leave, Dr. Singh was collecting,

managing, generating, and retaining research data in his laboratory. CP 903.

Consideration supported the University's unilateral contract in GIM 37 with Dr. Singh. Consideration may be established where employees continue in employment when they otherwise would not be required to do so, thus incurring a legal detriment. *Storti*, 181 Wn.2d at 37–38. Dr. Singh—like so many other faculty at the University—relied on the University's promises in GIM 37 by continuing to generate research data and by collecting, managing, generating, and retaining such data for the benefit of the University and of the greater scientific community.

At the very least, a jury could find the existence of a unilateral contract or specific promises based on the evidence Singh presented, and questions of whether employer policies and promises create contractual rights are generally fact questions that must be decided by a jury. *E.g.*, *Thompson*, 102 Wn.2d at 233 (reversing summary judgment in employer's favor due to

"material questions of fact" over employer promises); *Carlson*, 116 Wn. App. at 730 (accord); *see also Fenn v. Yale Univ.*, 283 F. Supp. 2d 615, 628–29 (D. Conn. 2003) (observing that university policies touching on intellectual property "have long been recognized as a valid and enforceable part of the contract of employment"). Division One overlooked the nature of these important factual and legal disputes, creating further conflicts in the law. RAP 13.4(b)(1), (2).

Absent formal employment agreements, faculty conducting research at any public-research institution are bound by the policies governing their employment relationship and intellectual property. But when these institutions disavow their obligations in those policies and point to the lack of a formal contract, the faculty are left with nothing to protect their interests in their scientific research, some of which has been created over decades. This Court should grant review and reverse because GIM 37 created a unilateral contract between the faculty and the University concerning rights and interests in research data.

B. A public-research institution's intentional destruction of a core research faculty's contractually protected research data is an issue of substantial public interest that warrants review.

Review is warranted because of the issues of substantial public importance presented by this case. RAP 13.4(b)(4). Although the principles of *Thompson* and *Storti* apply naturally here, this Court has never analyzed the promises, rights, and obligations reflected in university policy or so-called "guidance" documents, which may form a unilateral contract regarding faculty-generated research data and intellectual property. This important issue affects thousands of research faculty, not only at the University—one of the most prestigious research institutions in the world—but for faculty at the State's other prestigious institutions who have a property interest in their important research.

The "principal functions of a university are to preserve, to increase, and to transmit knowledge." CP 731 (the University's Faculty Code). Most major scientific breakthroughs in cancer

and vaccine research occur at public-research institutions like the University of Washington.[4]

Public-research institutions, such as the University of Washington, have historically played a major role in scientific and medical advances. *See supra*, n.4. Especially in the last few decades, intellectual property generated by faculty at these institutions has burgeoned. To accommodate the development of this property, the University enacted several policies to balance the rights and interests in intellectual property between it and the faculty.

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[4] *See, e.g.*, Penn Medicine News, *2022 Breakthrough Prize in Life Sciences Awarded to Penn Medicine mRNA Pioneers Drew Weissman and Katalin Karikó*, Sept. 9, 2021 (honoring two University of Pennslyvania professors for engineering modified RNA technology that enabled rapid development of effective COVID-19 vaccines); Ted Mitchell, *University research is key to COVID-19 breakthroughs, serving the public good*, THE HILL, March 23, 2020 (arguing that the "collective societal investment in institutions of higher learning pays huge dividends in scientific and medical advances, our country's overall economic prosperity and social well-being, and ensures a diverse and flourishing democracy").

The University has a "valid interest" but not the sole interest in all faculty discoveries, including Dr. Singh's. CP 1233. The bundle-of-sticks metaphor for traditional real-property rights aptly describes the protectable interests in research data granted respectively to the University and its faculty who generate the data. CP 902–03. The purpose of GIM 37 was to establish the "rights in research data" generated at the University. CP 902. These rights included ownership, stewardship, and preservation. CP 903–04. The principal investigator generally controlled access to their research data, and the research data could only be destroyed by the principal investigator. CP 903–05.

Under GIM 37, the principal investigator has "the ultimate responsibility for destruction of research data." CP 905. The University has no right under GIM 37, or under any other contract identified by the University, to destroy a core faculty member's research data. It must instead preserve and protect the research data—not destroy it—for the benefit of the principal

investigator, who must in exchange collect, manage, and retain the data. CP 903–05.

In addition, the University's right to take custody of research data under GIM 37 existed only "where necessary to assure needed and appropriate access." CP 904. But access is not equivalent to destruction. Nowhere in GIM 37 does it grant the right to the University to destroy a principal investigator's research data. And for good reason. That research data is vital for advancing scientific and medical cancer research. Even worse, the University's destruction of Dr. Singh's ARTN cell line conflicts with the very purpose of its existence: "to preserve, to increase, and to transmit knowledge" (CP 731), "to seek new knowledge for the general benefit" (CP 850), and "to preserve, protect, and share Research Data" (CP 902).

Singh identified that the University improperly destroyed her late husband's licensed ARTN cell line. GIM 37 did not give the University the right to destroy that cell line. CP 905. And Dr. Singh had a protectable interest in that cell line.

For these reasons, and because this breach-of-contract claims presents an issue of substantial public interest, review is warranted. Otherwise, if the Court of Appeals' decision is left to stand, a policy will be endorsed that permits the University, or any other university in Washington, to dismantle and to destroy a lifetime of DNA and cancer research by a core faculty who was an international leader in his field. Where the dissemination of knowledge is critical to public health, if anything, this global pandemic has proven that scientific discovery thrives only with collaboration and protection of research.

C. A public-research institution's unilateral decision to terminate a licensing agreement concerning a faculty's research data and intellectual property in which that faculty has a protectable interest, and later to destroy the property underlying that agreement, presents an issue of substantial public interest that warrants review.

Likewise, Division One's treatment of Singh's tortious-interference claim cannot stand, and this Court should review this issue of substantial public importance. RAP 13.4(b)(4).

Dr. Singh personally generated revenue from his intellectual property, and this revenue served to support his continued research. CP 844–45. Dr. Singh's ARTN cell line showed great potential, promising to support cancer research, particularly "cancer therapies, early disease identification, [and] drug targeting." CP 917.

During his medical leave, Dr. Singh completed a Record of Innovation Form to begin the process of patenting the cell line and potentially licensing it to the business sector. CP 916–21. He agreed to assist the University in commercializing the cell line in exchange for receiving licensing revenue. CP 920.

The University ultimately entered into a nonexclusive-license agreement with ABM and received a $1,000 deposit from ABM. CP 1450–55. Yet the University later unilaterally terminated it and destroyed the ARTN cell line.

The University's own actions prevented it from performing the ABM agreement—an agreement in which Dr.

Singh had a protectable interest. That protectable interest came in the form of a "prospective contractual or business relationship" of pecuniary value with ABM. *Manna Funding, LLC v. Kittitas County*, 173 Wn. App. 879, 897, 295 P.3d 1197 (2013).

The University terminated the ABM agreement, and thus interfered with Dr. Singh's business expectancy, through an improper purpose or means. The University refused to allow Dr. Singh's representatives, as well as other faculty and former laboratory assistants, to manage his laboratory during the medical leave. CP 2315, 2326. The University then used the lack of laboratory personnel to terminate the ABM agreement. CP 1581–83. The University itself made the cell lines of the ABM agreement inaccessible. CP 2315, 2326. The University covertly decommissioned Dr. Singh's laboratory when he was on medical leave. CP 783–84, 1406, 1552–53. And the University sought to delete evidence of Dr. Singh's pending grant because

it could prove "incriminating" in its efforts to decommission Dr. Singh's lab. CP 2322.

Dr. Singh imparted the knowledge and experience to locate and ship the ARTN cell line with appropriate culturing instructions to his students, his faculty colleagues, and other researchers through both published and internal protocols. *See Appellant's Mot. Recons.* 14 n.5. And when Dr. Singh's medical condition became serious, many volunteered to do so. The reasonable inference from the record is that both Dr. Pollack and one of Dr. Singh's former lab assistants could have located and shipped the cell lines with the appropriate culturing instructions to ABM. CP 1737, 2315, 2326. That evidence is corroborated by the letter Singh's trial counsel sent to the University in August 2016. CP 1461 ("[T]he University has decided that it is entitled to deny access to Dr. Gerald Pollack, another credentialed scientist in the BioEngineering Department, who has volunteered to maintain the laboratory and do what is

necessary to preserve and protect whatever cultures and experiments may be ongoing within the laboratory.").

Although a party cannot tortiously interfere with its own contract, a party can tortiously interfere with a contract in which another has a valid business expectancy. *E.g.*, *Newton Ins. Agency & Brokerage, Inc. v. Caledonian Ins. Grp., Inc.*, 114 Wn. App. 151, 157–58, 52 P.3d 30 (2002). The University and Dr. Singh shared in all royalties and licensing revenue from Dr. Singh's research data, including his cell lines. CP 852, 883, 900. So Dr. Singh had a business expectancy from the commercialization of his research data.

Review is warranted because hundreds of research faculty like Dr. Singh rely on the revenue derived from their intellectual property to support their salaries and research. Whether the University is entitled to unilaterally terminate a licensing agreement in which a core faculty member has a valid business expectancy presents an issue of substantial public interest that should be determined by this Court. RAP 13.4(b)(4).

D. Review is warranted because core research faculty's right to be paid wages for classroom instruction presents an issue of substantial public interest.

Persons employed in Washington have the right to be paid wages for their work. *See generally* chapter 49.46 RCW (Washington Minimum Wage Act); RCW 49.48.010 (requiring payment of wages due an employee ceasing work to be at end of pay period). So too do faculty employed at a public-research institution, including the University of Washington. Again, Division One was wrong to brush these important public issues aside by affirming the summary judgment and denying Singh her day in court. Review is warranted. RAP 13.4(b)(4).

Research faculty, as the name suggests, principally conduct research in laboratories within their respective colleges, schools, or departments. But research faculty also "may participate in the regular instructional program," even though they "are not required to do so[.]" CP 739. If they do so, "[c]lassroom instructional duties shall be supported from departmentally administered funds." CP 740; *see also* CP 1743–

44 (noting that research faculty are paid for their teaching activities).

Like so many other research faculty at the University generally and in its Bioengineering Department specifically, Dr. Singh both conducted research and taught classes. CP 1089–93. The University historically paid wages to Dr. Singh for his teaching and mentoring activities. CP 775. According to Dr. Singh's resume, which was produced by the University in discovery, Dr. Singh was currently teaching at least four bioengineering classes when he began an extended medical leave. CP 1300. The University's formula for calculating teaching wages was "[Course equivalent] X [Instructor's monthly full-time salary up to NIH salary cap]." CP 1511, 1745, 1874. Despite that Dr. Singh undisputedly performed teaching activities before his medical leave, the University failed to pay contractually and statutorily obligated wages to Dr. Singh for those activities.

Review is warranted because there are perhaps hundreds, if not thousands, of non-tenured research-like faculty employed at the University of Washington who both conduct research and perform classroom instruction. These faculty are integral to furthering the University's academic mission and to leading the next generation of scientific and medical scholars. They are not second-class faculty deserving of fewer rights and less protections compared to tenured-teaching faculty. This wage issue presents an issue of substantial public interest that should be determined by this Court.

VI. CONCLUSION

Division One's decision conflicts with precedent regarding unilateral-contractual rights for Washington employees. These issues involve a major State public-research institution and intellectual property, carrying significant implications for hundreds, if not thousands, of faculty conducting research across the State. Review is warranted under RAP 13.4(b)(1)–(2), (4).

This document contains 4,995 words, excluding the parts of the document exempted from the word count by RAP 18.17.

Respectfully submitted: November 18, 2021.

CARNEY BADLEY SPELLMAN, P.S.

By */s/ Rory D. Cosgrove*

Rory D. Cosgrove, WSBA No. 48647

Attorney for Petitioner Asha Singh, as the personal representative of the estate of her late husband, Narendra P. Singh

CERTIFICATE OF SERVICE

The undersigned certifies under penalty of perjury under the laws of the State of Washington that I am an employee at Carney Badley Spellman, P.S., over the age of 18 years, not a party to nor interested in the above-entitled action, and competent to be a witness herein. On the date stated below, I caused to be served a true and correct copy of the foregoing document on the below-listed attorneys of record by the method noted:

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DATED: November 18, 2021.

S/ Rozalynne Weinberg

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APPENDIX A

FILED
8/16/2021
Court of Appeals
Division I
State of Washington

IN THE COURT OF APPEALS OF THE STATE OF WASHINGTON

ASHA SINGH, personally and as Personal Representative of the Estate of NARENDRA P. SINGH, Appellants, v. STATE OF WASHINGTON, a Governmental entity; UNIVERSITY OF WASHINGTON, a Washington State entity; and JOHN DOES 1-5, Respondents.	)	No. 80662-9-I DIVISION ONE UNPUBLISHED OPINION

MANN, C.J. — Asha Singh, personally, and as a personal representative of the estate of her late husband, Dr. Narendra Singh, appeals an order granting summary judgment in favor of the University of Washington dismissing claims for: breach of contract; tortious interference with contract; breach of Washington's Wage and Hours Act (WWHA), ch. 49.46 RCW; conversion; and failure to accommodate under Washington's Law Against Discrimination (WLAD), ch. 49.60 RCW. Singh also challenges additional presummary judgment orders including: dismissal of claims brought under the Public Records Act (PRA), ch. 42.56 RCW; an award of sanctions for failing to attend depositions; denial of continuance requests; and the exclusion of an expert witness.

Because there is a genuine issue of material fact regarding the reasonable accommodation claim, we reverse summary judgment and remand for trial on that claim alone. We otherwise affirm.

FACTS

A. Background

From 1998 to 2016, Dr. Narendra Singh worked as research faculty for the University of Washington's Department of Bioengineering (Department). Dr. Singh's primary responsibility as research faculty was to conduct research. For 25 years, Dr. Singh conducted groundbreaking work on DNA[1] damage, including developing a technique known as the "comet assay" to quantify DNA damage at the cellular level. Dr. Singh was not tenured and did not receive a salary directly from the University. Instead he was required to obtain grant funding to cover his base salary and research.

In April 2012, Dr. Singh asked the University to reduce his appointment to 50 percent full-time equivalent (FTE) given his low level of research funding and activity. The University approved the request. The following year the University provided bridge funding because Dr. Singh had not generated sufficient funding to cover his base salary. Still unable to pay himself when the bridge funding ended in 2014, the Department voluntarily paid 50 percent of Dr. Singh's salary, but advised him that his faculty employment might end if he did not obtain research funding.

Dr. Singh secured a grant for research concerning "Mobile Phone Use and DNA damage." The grant provided the minimum funding that the University required from

[1] Deoxyribonucleic acid.

approximately January 1, 2015, until September 18, 2015. When it ended, Dr. Singh had no funding.

Dr. Singh was diagnosed with Parkinson's disease in 2003. In 2007, following an accident where Dr. Singh injured his hand with liquid nitrogen, Department Administrator Ruth Woods (Woods) completed a disability request form on Dr. Singh's behalf for an "hourly assistant to help in [the] office and laboratory." Woods submitted the form to the University's Disability Services Office ("DSO"), which handles disability accommodation requests for the University. The DSO requested Dr. Singh produce a healthcare provider statement to support the accommodation, which Dr. Singh never provided. The University nonetheless provided Dr. Singh with a student assistant on three separate occasions until September of 2015, when it ceased to do so.

On January 13, 2016, Dr. Singh collapsed at home and was taken to Swedish Hospital. Dr. Singh's wife, Asha Singh, advised Woods that Dr. Singh was "quite ill" and hospitalized but did not provide further detail. The University voluntarily changed Dr. Singh's FTE status and voluntarily paid him so that he received medical benefits retroactive to October 2015. The following month, Singh advised Woods that Dr. Singh was still comatose and requested family medical leave for him, initially for a series of months, and then through January 13, 2017, a full year after the incident. The University approved the leave.

Dr. Singh's family had him discharged and transported to India in February 2016. Dr. Singh passed away in India on December 2, 2016. It is undisputed that Dr. Singh was unable to work or conduct research after January 13, 2016.

In early 2016, while Dr. Singh was comatose in India, his adult daughter, Himani,[2] used Dr. Singh's University e-mail account and sent a series of e-mails to a private company called Applied Biological Materials (ABM). In the e-mails, Himani expressed Dr. Singh's interest in licensing to ABM a cell line known as "RTN."[3] Himani did not disclose her identity or that her father was comatose in India.

Himani then submitted to the University's CoMotion Department, which manages University intellectual property, paperwork to allow the University to commercialize the RTN cell line and begin licensing negotiations with ABM. Himani completed the "Record of Innovation and Assignment Form" in which she described, using the first person, how Dr. Singh developed the cell line while working at the University with the University's bridge funding. She applied Dr. Singh's electronic signature to the document, and in doing so, affirmatively represented that Dr. Singh had assigned all rights to the RTN cell line to the University and further warranted that Dr. Singh would assist the University in evaluation and possible commercialization of the line.

Unaware of Dr. Singh's incapacity or the forged paperwork, CoMotion negotiated and finalized a licensing agreement with ABM in October 2016. In November and December 2016, unaware of Dr. Singh's condition and subsequent passing, CoMotion

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[2] We refer to Dr. Singh's children by their first names in order to avoid confusion. We mean no disrespect.

[3] For example, in one e-mail Himani wrote:

> I apologize for the delay in responding to your email and I thank you for getting in touch. Developing the RTN cell line was the basis for my interest in cancer stem cells and studying chemotherapy resistance. I would be interested in collaborating with you and furthering this endeavor. Please let me know how you would like to proceed.
> . . .
> Narendra P. Singh

repeatedly requested Dr. Singh ship materials to ABM, as well as the technical information required to culture and maintain the cells. No one responded.

In March 2017, CoMotion learned of Dr. Singh's December 2016 passing. Because no one had the technical knowledge to culture the RTN cells, CoMotion terminated the agreement with ABM and refunded an upfront down payment of one thousand dollars before any licensing fees were obtained.

On January 17, 2017, attorney Laruen Parris Watts, purporting to represent the Singh family, informed University counsel that Dr. Singh had passed away. Watts requested that Singh be allowed to obtain Dr. Singh's personal items that he had purchased with his own money. The University requested the Singh family provide documentation showing Dr. Singh's ownership of any particular items. On February 14, 2017, the University provided the family with some boxes of Dr. Singh's personal items. On June 16, 2017, following cataloguing of records and identification of additional items, the University provided the Singh family with more than 40 boxes of additional items that appeared were Dr. Singh's personal property.

B. Procedural History

On September 14, 2018, Singh, both in her personal capacity and as a personal representative of Dr. Singh's estate, commenced this action. The complaint alleged intentional and negligent infliction of emotional distress, breach of contract, tortious interference with business expectancy, failure to pay wages, failure to accommodate a disability, and trespass to chattels/conversion. The case scheduling order set a July 2019 discovery deadline with a September 2019 trial date. Singh filed an amended

complaint in June 2019, adding claims for violations of the PRA and wrongful termination.

On September 3, 2019, the trial court granted the University's motion for summary judgment dismissing all of Singh's claims.

Singh appeals.

ANALYSIS

A. Claims Dismissed on Summary Judgment

Singh first contends that the trial court erred in granting summary judgment and dismissing claims for: breach of contract; tortious interference with contract; breach of WWHA; conversion; and failure to accommodate under WLAD. We address each claim in turn.

We review summary judgment decisions de novo. Int'l Marine Underwriters v. ABCD Marine, LLC, 179 Wn.2d 274, 281, 313 P.3d 395 (2013). "Summary judgment is proper only where there is no genuine issue of material fact and the moving party is entitled to judgment as a matter of law." Int'l Marine Underwriters, 179 Wn.2d at 281. The moving party has the initial burden of proving the absence of an issue of material fact. Young v. Key Pharms., Inc., 112 Wn.2d 216, 225, 770 P.2d 182 (1989). If the moving party successfully carries that burden, the burden then shifts to the nonmoving party to set forth specific facts rebutting the moving party's contentions and showing that a genuine issue of material fact exists for trial. Pac. Nw. Shooting Park Ass'n v. City of Sequim, 158 Wn.2d 342, 351, 144 P.3d 276 (2006). The party opposing summary judgment may not rely on speculation or argumentative assertions that

unresolved factual issues remain. Seven Gables Corp. v. MGM/UA Entm't Co., 106 Wn.2d 1, 13, 721 P.2d 1 (1986).

1. Breach of Contract

In order to demonstrate a breach of contract, a plaintiff must demonstrate that "a valid agreement existed between the parties, the agreement was breached, and the plaintiff was damaged." Univ. of Wash. v. Gov't Emps. Ins. Co., 200 Wn. App. 455, 467, 404 P.3d 559 (2017). Singh avers that the trial court erred in granting summary judgment because there was evidence in the record that the University breached its contract with Dr. Singh by failing to preserve his work and administer licensing agreements for his intellectual property. We disagree.

Singh first contends that the University breached the contract by destroying Dr. Singh's work. The University counters that Singh has not identified any work that was destroyed and that, regardless, the University owns Dr. Singh's research data. The University is correct.

Singh relies largely on the University Grants Information Management Memorandum 37 (GIM 37) for the proposition that the University was obligated to "ensur[e] and protect[] the proper management of research data under the initial authority of the Principle Investigator." While this may be true, GIM 37 is a guidance document that provides "principles regarding rights in Research Data" and must be read "in conjunction with applicable laws, contract terms, and University policies." Additionally, GIM 37 is clear that "Research Data" belongs to the University, and that while Dr. Singh may have generally determined who has access to his data, "where

necessary to assure needed and appropriate access, the University has the option to take custody of any or all research data."

Singh next argues that the University breached a contract when it failed to pay Dr. Singh royalties, and that it was required to take additional steps to seek third-party agreements involving Dr. Singh's research. The University counters that it acted in conformity with the documents governing Dr. Singh's research materials, and that it had no obligation to seek additional assistance in maintaining Dr. Singh's cell lines or further licensing them. The University is correct.

After Dr. Singh's incapacitation, he was unable to answer questions regarding his research. Singh produced no admissible evidence that any faculty member was available or qualified to continue the research in Dr. Singh's absence.[4] In addition, Singh has cited no contractual provision requiring the University to allow another faculty member or member of Dr. Singh's family to care for Singh's research data. Finally, per the University's Patent, Invention, and Copyright Policy (PIC Policy), Dr. Singh's assignment of the cell line, and GIM 37, the University owned Dr. Singh's research material, which it had the right to determine how to handle.

Further, following Dr. Singh's incapacitation and subsequent passing, the University was unable to perform under the obligations of its licensing agreement with ABM. Dr. Singh was the only person with the unique knowledge and experience to ship the cell line with the necessary culturing instructions. Thus, the University could not receive royalty payments from ABM and could not provide Dr. Singh his portion of the

[4] The only evidence offered by Singh is a letter by one of plaintiff's attorneys asserting that Dr. Gerald Pollack was qualified and had volunteered to maintain Dr. Singh's lab. We agree with the trial court that this assertion is hearsay and inadmissible to defeat summary judgment.

payments. And the University was not required to seek additional buyers for Dr. Singh's research materials; Singh provides no evidence to the contrary. The University had no implied obligation to use reasonable efforts to find a buyer absent a legal necessity to do so. Oliver v. Flow Intern. Corp, 137 Wn. App. 655, 661, 155 P.2d 140 (2006).

Finally, Singh argues that the University breached an implied duty of good faith and fair dealing. "There is in every contract an implied duty of good faith and fair dealing [that] obligates the parties to cooperate with each other so that each may obtain the full benefit of performance." Badgett v. Sec. State Bank, 116 Wn.2d 563, 569, 807 P.2d 356 (1991). This duty to cooperate, however "exists only in relation to performance of a specific contract term." Badgett, 116 Wn.2d at 570.

The University complied with any type of implied good faith and fair dealing. CoMotion exhausted resources to come to an agreement with ABM, an agreement that was initially created when Himani posed as Dr. Singh. CoMotion attempted to perform on the contract and deliver the RTN cell line. It was only after learning of Dr. Singh's passing that it terminated the licensing agreement and refunded ABM's $1,000 initial payment. If the University could not perform the contract, then Dr. Singh was entitled to no proceeds—including the initial down payment.

There is no genuine issue of material fact regarding a breach of contract between Dr. Singh and the University. Summary judgment and dismissal of the breach of contract claims was appropriate.

2. Wage and Hours Act

Singh next argues that genuine issues of material fact barred summary judgment on his claim that the University failed to pay Dr. Singh his teaching wages, and its failure

to pay violated WWHA. Singh failed, however, to raise this claim in the complaint—instead raising it for the first time in response to the University's motion for summary judgment. "Complaints that fail to give the opposing party fair notice of the claim asserted are insufficient." Pac. Nw. Shooing Park Ass'n, 158 Wn.2d at 352. Singh's teaching wage claim is not properly before us and we decline to address this issue. Pac. Nw. Shooing Park Ass'n, 158 Wn.2d at 353.[5]

3. Tortious Interference

Tortious interference with a business expectancy arises when there is (1) a valid business expectancy; (2) the defendant had knowledge of that expectancy; (3) the defendant intentionally interfered, causing termination of the expectancy; (4) the interference was or an improper purpose or used improper means; and (5) the plaintiff was damaged as a result. Pac. Nw. Shooting Park Ass'n, 158 Wn. 2d at 351-52. Singh argues that genuine disputed of material fact barred summary judgment against the claim for tortious interference of business expectancy. We disagree.

Singh failed to establish the elements of a tortious interference claim. For the license agreement with ABM, Dr. Singh's incapacitation and subsequent passing prevented the University from fulfilling its obligation under the agreement. Understanding that Dr. Singh was the only person with the unique knowledge and experience to locate and ship the RTN cell line with appropriate culturing instructions, CoMotion e-mailed Dr. Singh four times requesting he ship the required materials. No

[5] Singh argues in her Reply that the discovery rule should apply because she was only notified of the teaching wages formula in June 2019. The discovery rule, however, only tolls the date of accrual until the plaintiff "knows or, through the exercise of due diligence, should have known all the facts necessary to establish a legal claim." Crisman v. Crisman, 85 Wn. App. 15, 20, 931 P.2d 163 (1997). Here, Singh's delay in obtaining discovery demonstrates that she did not exercise due diligence, thus the discovery rule does not apply.

one responded. Once CoMotion learned of Dr. Singh's passing, ABM and the University mutually terminated the agreement. The University had an unfettered right to "contract management" regarding the RTN cell line.

Further, nothing in the record suggests that the University terminated the contract through an improper purpose, or an improper means. "Exercising in good faith one's legal interests is not improper interference." Leingang v. Pierce County Med. Bureau, Inc., 131 Wn.2d 133, 157, 930 P.2d 288 (1997). No one was available to culture and care for the licensed cell line, therefore the University and ABM mutually terminated their agreement.

Additionally, Singh cannot assert her tortious interference claim on the ABM-University agreement. "A party cannot tortuously interfere with its own contract." Reninger v. Dep't of Corrections, 134 Wn.2d 437 448, 951 P.2d 782 (1998).

Singh cites Cherberg v. Peoples Nat. Bank of Washington, 88 Wn.2d 595, 603, 564 P.2d 1137 (1977), for the premise that "in those instances in which the conduct of the breaching party indicates a motive to destroy some interest of the adverse party, a tort action may lie and items of damage not available in contract actions will be allowed." Here, however, Singh has cited no evidence that the University had a motive to destroy interests of Dr. Singh. Rather, the record reflects that the University attempted to fulfill the contract with ABM and that, upon learning of Dr. Singh's passing, the University and ABM mutually terminated the contract.

There is no evidence on record that the University interfered with Dr. Singh's business expectancy. Similar to the pleadings at the trial court, Singh's claims revolve around unsupported speculation. Singh lacks evidence that anyone could have cared

for the cell lines. Further, the cell lines and instructions for their care were never sent to ABM and the University did not dissolve the contract until learning of Dr. Singh's passing and concluding that his expertise alone could provide the cell lines to ABM. These facts combined do not rise to tortious interference by the University.

4. Conversion

Singh argues genuine issues of material fact barred summary judgment as to the University's conversion of Dr. Singh's property. Singh contends there remain factual dispute over whether (1) Dr. Singh owned the research data in his laboratory; (2) the University was privileged to destroy hazardous chemicals, and dispose of biological materials; and (3) there were personal items in Dr. Singh's laboratory that belonged to him.

"Conversion is the unjustified, willful interference with a chattel which deprives a person entitled to the property of possession. The burden is on the plaintiff to establish ownership and a right to possession of the converted property." Meyers Way Dev. Ltd. Partnership v. Univ. Savings Bank, 80 Wn. App. 655, 674-75, 910 P.2d 1308 (1996). At a minimum, a plaintiff must establish that they have "some property interest in the goods allegedly converted." Meyers Way Dev. Ltd. Partnership, 80 Wn. App. at 675 (internal quotations and citations omitted). Additionally, "[o]ne is privileged to commit and act which would otherwise be a . . . conversion if [they are] acting in discharge of a duty or authority created by law to preserve the public safety, health, peace, or other public interest, and [their] act is reasonably necessary to the performance of [their] duty or the exercise of [their] authority." RESTATEMENT (SECOND) OF TORTS § 265 (AM. LAW INST. 1965).

Cell Lines

With respect to Dr. Singh's cell lines and research data that he developed at the University, the University correctly points out that Dr. Singh failed to demonstrate ownership. Dr. Singh agreed to assign all inventions and discoveries that he made while a University employee to the University and further agreed that the University "shall own and hold and such patents, trademarks, or copyrights emanating from those discoveries and inventions." The PIC Policy binding Dr. Singh provided that "[a]s a condition of employment . . . University employees agree to assign all inventions in which the University has an interest to the University." GIM 37 states "[a]ll research data[6] is owned by the University, except as otherwise provided by an agreement with a third party, a law, or University policy, such as copyright policy."

Collectively, these documents bound Dr. Singh. He developed the RTN cell line in 2014 through University provided bridge funding, and subsequently assigned all rights to the cell line to the University through the Record of Innovation and Assignment. Singh failed to demonstrate that there remained a material fact over ownership of the cell lines.

Chemicals and biologic materials

Singh contends that the University had a duty to maintain biological materials in Dr. Singh's laboratory. As the University correctly explains, however, once Dr. Singh became incapacitated and later died, the University was required to dispose of biological waste remaining in the laboratory. University policy requires its personnel to regulate and properly handle all hazardous and biological/infectious waste materials

[6] GIM 37's definition of "research data" includes "cell lines."

under state hazardous waste laws and regulations. Under University policy, "biohazardous waste" includes "cell lines" and "specimen cultures and cultures." Generally, individual researchers "and/or departmental managers/supervisors are responsible for identifying biohazardous waste generated by their activity." After Dr. Singh's death, the responsibility fell to the Department.

The record demonstrates that, following Dr. Singh's passing, the Department disposed of the biological materials in his lab consistent with the University's Environmental Safety & Health Guidelines, thus fulfilling its obligation to dispose of hazardous waste. This was not an unjustified or willful action interfering with Dr. Singh's property.

Other Personal Materials

Singh argues finally, that there was an "insufficient record of what items in Dr. Singh's lab belonged to him" and therefore an issue remained for trial over whether the Department converted Dr. Singh's personal items.

In response to the Department's motion for summary judgment, Singh submitted a declaration stating that she recalled Dr. Singh "driving a U-haul truck containing the contents of his USC laboratory to the Health Sciences Building at the University of Washington . . . [and] numerous charges for scientific equipment and supplies on our personal debit cards and checks written from our joint account." Singh also offered a statement from one of Dr. Singh's colleagues, Dr. Gerhardt, stated "Dr. Singh's devotion to his work included the purchase of equipment by his own expense."

The Department offered the declaration of its records manager, Elizabeth Mounce. Mounce explained that in January 2017, after learning Dr. Singh had died and

that his family requested the contents of his office and lab, she cataloged the office to identify materials that could be provided and in February 2017 released all items from his office that were "readily identifiable as his personal belongings." Mounce further explained:

> I understand that in March 2017, Plaintiff Asha Singh, through an attorney, claimed that Dr. Singh had used personal funds to purchase equipment for use in his laboratory, and that documentation of those purchases was stored in Dr. Singh's office at the University. As part of the ongoing cataloging process, I made note of all receipts for purchases, to ensure those materials would be provided to Plaintiff. I completed the cataloging process in early June 2017.
>
> On June 16, 2017, the University released to Asha Singh over 40 banker's boxes of materials from Dr. Singh's office, including any and all personal receipts, invoices, and bank statements that were kept there, as well as copies of all receipts for purchases made with University funds. Asha Singh, Himani Singh, and Arun Singh retrieved the boxes on June 16, 2017, and I supervised the retrieval.

Other than the speculation that there may be additional materials, Singh fails to offer evidence, such as receipts or credit card statements, to create a material issue of fact. Dismissal of Singh's claims for conversion was appropriate.

F. Failure to Reasonably Accommodate under the WLAD

Singh argues that the trial court erred by dismissing Dr. Singh's claim for failure to accommodate under the WLAD.[7] We agree.

Claims under the WLAD are typically inappropriate for resolution at summary judgment "because the WLAD 'mandates liberal construction' and the evidence 'will generally contain reasonable but competing inferences of both discrimination and nondiscrimination that must be resolved by a jury.'" Johnson v. Chevron U.S.A., Inc.,

[7] Singh's accommodation claim is limited to the University's discontinuance of a student "support for assistance" in September 2015, as any earlier claims would be barred by the statute of limitations. RCW 4.16.080(2); Antonius v. King County, 153 Wn.2d 256, 261-62, 103 P.3d 256 (2004).

159 Wn. App. 18, 27, 244 P.2d 438 (2010) (footnote omitted) (quoting Martini v. Boeing Co., 137 Wn.2d 357, 364, 971 P.2d 45 (1999)); Davis v. W. One Auto Grp., 140 Wn. App. 449, 456, 166 P.2d 807 (2007). We only "grant summary judgment when the plaintiff fails to raise a genuine issue of fact on one or more prima facie elements." Johnson, 159 Wn. App.at 27.

The WLAD prohibits an employer from discriminating against any person because of "the presence of any sensory, mental, or physical disability" RCW 49.60.180(3), and supplies a cause of action "when the employer fails to take steps reasonably necessary to accommodate an employee's" disability. Johnson, 159 Wn. App. at 27; RCW 49.60.030(1)(a). Remedies for a violation of the WLAD include injunctive relief, actual damages, and costs, including reasonable attorney fees. RCW 49.60.030(2).

The WLAD defines disability as "the presence of a sensory, mental, or physical impairment that: (i) is medically cognizable . . .; or (ii) [e]xists as a record or history; or (iii) [i]s perceived to exist." RCW 49.60.040. There is no dispute that Dr. Singh's Parkinson's disease was a qualifying limitation under the WLAD.

"[T]o establish a prima facie case of failure to reasonably accommodate a disability" under the WLAD, "a plaintiff must show that (1) the employee had a sensory, mental, or physical abnormality that substantially limited his or her ability to perform the job; (2) the employee was qualified to perform the essential functions of the job in question; (3) the employee gave the employer notice of the abnormality and its accompanying substantial limitations; and (4) upon notice, the employer failed to affirmatively adopt measures that were available to the employer and medically

necessary to accommodate the abnormality" Davis v. Microsoft Corp., 149 Wn.2d 521, 532, 70 P.2d 126 (2003); RCW 49.60.040(7).

When an employer has notice of a disability, "this notice then triggers the employer's burden to take 'positive steps' to accommodate the employee's limitations." Goodman v. Boeing Co. 127 Wn.2d 401, 408, 899 P.2d 1265 (1995). Employers must take these steps unless it "would impose an undue hardship on the conduct of the employer's business." Doe v. Boeing Co. 121 Wn.2d 8, 18, 846 P.2d 531 (1993). The onus is on the employee to "giv[e] the employer notice of the disability." Goodman, 127 Wn.2d at 408. Once notice is given, the employee also "retains a duty to cooperate with the employer's efforts . . . [The WLAD] thus envisions an exchange between employer and employee where each seeks and shares information to achieve the best" possible outcome. Goodman, 127 Wn.2d at 408-09.

It is undisputed that the University knew of Dr. Singh's Parkinson's disease. There is a genuine dispute however, whether providing Dr. Singh with research assistants was an affirmatively adopted measure necessary to accommodate Dr. Singh's disability.

After Dr. Singh burned his fingertips with liquid nitrogen, Woods submitted a disability accommodation request form on Dr. Singh's behalf for an "hourly assistant to help in [the] office and laboratory." In her declaration, however, Woods claims that student assistants later supplied to Dr. Singh were provided as a means of support for faculty who were struggling to obtain funding. Two of Dr. Singh's former student assistants submitted declarations describing tasks they performed as assisting Dr. Singh with limitations involving his Parkinson's including helping him up from his chair,

helping him place the key in his office door, giving presentations due to his vocal difficulties, and helping him with anything that his tremors prevented him from doing easily.

In 2015, the University provided Dr. Singh with research assistant K.V. Dr. Singh wrote that K.V. assumed "increased tasks and responsibilities that include helping [him] write grants and manuscript preparation," thus implying that K.V. assisted Dr. Singh due to his physical limitations. The University counters that K.V. worked for the traditional research assistance that faculty seek and not as an accommodation for Dr. Singh. Whether K.V. was provided as an accommodation is a material dispute.

The University responds that Dr. Singh failed to notify it of an ongoing need for accommodation. The University cites Gamble v. City of Seattle, 6 Wn. App. 2d 883, 893-95, 431 P.3d 1091 (2018), for the proposition that Dr. Singh had a continuing burden to inform the University that he required an assistant as an accommodation. In Gamble, the employer accommodated its employee in a number of ways, including a "four ten" schedule. The employer subsequently changed its policy to disallow "four ten" schedules. Gamble, 6 Wn. App. 2d at 893-95. When the employee returned from medical leave, she recognized the policy change and requested alternating Wednesday's off, which was granted. Gamble, 6 Wn. App. 2d at 893-95. On summary judgment, the trial court rejected the employee's argument that removing the "four ten schedule constituted a failure to accommodate; it was the employee's affirmative burden to inform the employer that she required the accommodation." Gamble, 6 Wn. App. 2d at 887, 895.

Here, unlike Gamble, Dr. Singh had an ongoing, debilitative, and worsening disease. The University provided Dr. Singh with an assistant from 2007 to 2015. If in fact these assistants were provided to assist Dr. Singh with his limitations due to Parkinson's, it makes no sense that Dr. Singh would need to notify the University for the University to know that he would always need someone to carry out tasks that his disability permanently prevented him from doing. Such is the nature of his disease. This raises two questions of material fact. First, if the research assistants were an accommodation for Dr. Singh's Parkinson's, was the termination of such assistance a violation of the WLAD? And second, if the research assistants were not an accommodation for Dr. Singh's Parkinson's, was there a complete failure to accommodate? Dismissal of Dr. Singh's accommodation claim under the WLAD was not appropriate.

B. Other Trial Court Orders

Singh also challenges several presummary judgment orders including: dismissal of claims brought under the PRA; an award of sanctions for failing to attend depositions; denial of continuance requests; and the exclusion of an expert witness. We address each in turn.

1. Public Records Act

Singh argues that the trial court erred in dismissing her claim for PRA violations under CR 12(b)(6). We disagree. Dismissal of a claim under CR 12(b)(6) is a question of law this court reviews de novo. Daniels v. State Farm Mut. Auto. Ins. Co., 193 Wn.2d 563, 571, 444 P.3d 582 (2019).

On January 27, 2017, Singh requested documents from the University under the PRA. The University produced two batches of records on May 3 and June 17, 2017. The latter installment included a letter stating "this concludes the University's response to your public records request." Two years later, on June 4, 2019, Singh filed their first amended complaint adding a PRA claim and alleging that the University failed to make an "adequate search" for responsive records.

Claims for an alleged violation of the PRA "must be filed within one year of the agency's claim of exemption or the last production of a record on a partial or installment basis." RCW 42.56.550(6). This "statute of limitations begin[s] on an agency's final, definitive response to a public records request." Belenski v. Jefferson County, 186 Wn.2d 452, 460, 378 P.3d 176 (2016). There is no dispute that Singh's PRA claim was filed more than one year after the University closed its response to Singh's record request.

Singh argues that the trial court erred in failing to apply equitable tolling to allow the claim to proceed. Equitable tolling may apply to an otherwise time-barred PRA claim. Belenski, 186 Wn.2d at 458-59, 461-62. A party asserting equitable tolling bears the burden of pleading and ultimately proving "bad faith, deception, or false assurances by the defendant and the exercise of diligence by the plaintiff." Price v. Gonzalez, 4 Wn. App. 2d 67, 75-76, 419 P.3d 858 (2018). Here, Singh did not allege that the University engaged in bad faith, deception or false assurances. Rather, she alleges only that the documents produced in discovery "should have been identified and produced in response the original records request." Because Singh did not meet her

burden, equitable tolling does not apply and dismissal of the PRA claim was appropriate.

2. Sanctions for Failing to Attend Depositions

Singh argues that the trial court erred in sanctioning the Singhs after they did not attend their depositions. We disagree. We review an award of sanctions for an abuse of discretion. Wash. State Physicians Ins. Exch. & Ass'n v. Fisons Corp., 122 Wn.2d 299, 338, 858 P.2d 1054 (1993).

Under CR 37(d)(1), a trial court may impose sanctions against any party who fails to "appear before the officer who is to take his or her deposition, after being served with proper notice." Under CR 37(d)(3), "the court shall require the party failing to act or the attorney advising the party or both to pay the reasonable expenses, including attorney fees, caused by the failure, unless the court finds that the failure was substantially justified or that other circumstances make an award of expenses unjust."

In March 2019, counsel for the parties agreed on deposition dates in early May for Asha, Himani and, Arun. The University confirmed the deposition dates by issuing subpoenas. Three court days before the scheduled depositions, Singh's counsel proposed postponing the Singhs' depositions. After the University declined, Singh moved to shorten time and postpone the depositions, arguing that documents recently produced in discovery might prompt a motion for leave to amend. Singh's counsel then disclosed that they would be seeking leave to amend the complaint to dismiss the claim of negligent infliction of emotional distress and add a claim of retaliation and requesting a trial continuance. Singh's counsel also stated the Singhs family would not be attending the depositions. The Singhs did not appear for their depositions.

The University moved to dismiss pursuant to CR 43(f)(3), and alternatively, for attorney fees the University incurred due to the Singhs' failures to attend. The trial court denied the motion to dismiss, but ordered that Singh reimburse the University $13,295.96 for fees associated with the missed depositions, including the motion to dismiss, as well as the oppositions to Singh's motions to postpone and shorten time.

Here, the Singhs were obligated to attend their depositions. When they failed to do so, the trial exercised its authority under CR 37 to award attorney fees and costs, the trial court did not abuse its discretion.

3. Denial of Continuances

Singh argues that the trial court erred in denying her requests for continuances at trial. We disagree.

We review a decision to grant or deny a continuance for an abuse of discretion. Balandzich v. Demeroto, 10 Wn. App. 718, 720, 519 P.2d 994 (1974). A trial court may exercise its discretion to "manage its own affairs so as to achieve the orderly and expeditious disposition of cases." Woodhead v. Disc. Waterbeds, Inc., 78 Wn. App. 125, 129, 896 P.2d 66 (1995). A trial court may properly deny a motion for a continuance when "(1) the requesting party does not offer a good reason for the delay in obtaining the desired evidence; (2) the requesting party does not state what evidence would be established through the additional discovery; or (3) the desired evidence will not raise a genuine issue of material fact." Cameron v. Atlantic Richfield Co., 8 Wn. App. 2d 795, 812-13, 442 P.3d 31 (2019).

The original scheduling order set trial for September 16, 2019. After five months of discovery, Singh requested a six-month trial continuance and a stay of proceedings in

order to find new counsel. On May 8, 2019, the trial court granted a two-week stay but denied the continuance because "[i]t is unclear at this point whether Plaintiff will retain new counsel and, if so, whether that new counsel will actually need a continuance."

On May 10, 2019, Attorney Gail Luhn appeared as Singh's new counsel. Because of an upcoming surgery and the complexity of the case, Luhn filed a motion for a six-month trial continuance and for leave to file an amended complaint. The motion for leave to amend sought to add claims of interference with protected leave under the WLAD, alleged PRA violations, and for wrongful termination on account of disability. The motion also sought to withdraw Singh's personal claim for negligent infliction of emotional distress.

The trial court denied the six-month extension, but instead granted a five-week extension, which rescheduled the trial to October 21, 2019. The trial court also granted Singh's motion for leave to amend the complaint. Six weeks later, on July 15, 2019, Luhn withdrew and attorney David Adler filed a notice of appearance. Singh then filed a third motion for a four-month trial continuance. Adler had previously represented the Singhs on the same claims. Trial was still 12 weeks away. The trial court denied the trial continuance.

Based on the record before us, the trial court did not abuse its discretion in denying the requested trial continuances.

4. Exclusion of Expert Witness

Singh argues that the trial court erred in excluding her expert, Dr. Rick Schwartz, as a potential expert witness for trial and awarding the University its attorney fees for moving to exclude Schwartz. We disagree. We review decisions of the trial court to

admit or exclude evidence, as well as its award of sanctions, for an abuse of discretion. State v. Olmedo, 112 Wn. App. 525, 530, 49 P.3d 960 (2002); Wash. State Physicians Ins. Exch. &, 122 Wn.2d at 338.

On July 1, 2019, Singh untimely disclosed Dr. Rick Schwartz as a potential expert witness. The University filed a motion to exclude Dr. Schwartz because the disclosure was untimely and did not include a summary of his opinions or their basis, violating King County Local Civil Rule (KCLCR) 26(k)(3)(C).

After considering less severe sanctions under Burnet v. Spokane Ambulance, 131 Wn.2d 484, 933 P.2d 1036 (1979), on July 29, 2019, the trial court denied the motion to exclude Schwartz's testimony. The trial court instead required Singh to provide by noon on August 5, 2019, "a complete disclosure" regarding Schwartz, which includes all information required by KCLCR 26(k). The trial court stated that "failure to comply with this disclosure may result in further sanctions, including but not limited to the exclusion of Schwartz."

On August 5, 2019, Singh filed a motion for reconsideration on the trial court's order regarding Schwartz that included an unsigned declaration that lacked a summary of Schwartz's opinions or basis thereof. The University renewed its motion to exclude Schwartz, and requested the trial court order Singh to reimburse the University's attorney fees and costs associated with Singh's failure to timely and properly identify Schwartz. The trial court granted the motion and ordered Singh to pay the attorney fees and costs.

Singh asserts that, after the trial court's order granting summary judgment, the University's renewed motion for exclusion and sanctions became moot. Versuslaw, Inc.

v. Stoel Rives, LLP, 127 Wn. App. 309, 331, 111 P.3d 886 (2005). We disagree. While the motion for exclusion may be moot, the University's motion for sanctions is not. The University still had a legally cognizable interest in compensation for unnecessary efforts due to Singh's failure to follow the trial court's instructions.

The trial court denied the University's initial motion for exclusion, giving Singh a second chance to have Schwartz as an expert witness. The court instructed Singh to include a summary of Schwartz's opinions and a basis for them. She failed to follow this instruction. The trial court did not abuse its discretion.

We reverse summary judgment dismissal of Singh's claim for accommodation and remand for further proceedings. We otherwise affirm.

Mann, C.J.

WE CONCUR:

Appelwick, J.

APPENDIX B

IN THE COURT OF APPEALS OF THE STATE OF WASHINGTON

ASHA SINGH, personally and as Personal Representative of the Estate of NARENDRA P. SINGH, Appellants, v. STATE OF WASHINGTON, a Governmental entity; UNIVERSITY OF WASHINGTON, a Washington State entity; and JOHN DOES 1-5, Respondents.	)	No. 80662-9-I DIVISION ONE ORDER DENYING MOTION FOR RECONSIDERATION

Appellant Asha Singh moved to reconsider the court's opinion filed on August 16, 2021. Respondent University of Washington filed an answer. The panel has determined that the motion for reconsideration should be denied.

Therefore, it is

ORDERED that the motion for reconsideration is denied.

FOR THE COURT:

Mann, C.J.

APPENDIX C

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Reflections in Mutation Research

The comet assay: Reflections on its development, evolution and applications☆

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ABSTRACT

The study of DNA damage and its repair is critical to our understanding of human aging and cancer. This review reflects on the development of a simple technique, now known as the comet assay, to study the accumulation of DNA damage and its repair. It describes my journey into aging research and the need for a method that sensitively quantifies DNA damage on a cell-by-cell basis and on a day-by-day basis. My inspirations, obstacles and successes on the path to developing this assay and improving its reliability and sensitivity are discussed. Recent modifications, applications, and the process of standardizing the technique are also described. What was once untried and unknown has become a technique used around the world for understanding and monitoring DNA damage. The comet assay's use has grown exponentially in the new millennium, as emphasis on studying biological phenomena at the single-cell level has increased. I and others have applied the technique across cell types (including germ cells) and species (including bacteria). As it enters new realms and gains clinical relevance, the comet assay may very well illuminate human aging and its prevention.

1. Introduction

By the time I began research, it was already accepted that to understand the process of aging and its causes, one had to see DNA. The scientists who laid the foundations for our field were scientists who found a way to see: whether Sutton [1] and Boveri who saw that genes had to be located on chromosomes, Franklin and Gosling [2] who saw the structure of DNA, or Tjio and Levan [3] who saw the true number of human chromosomes. The desire to see human aging with as much clarity as I could was always my main mission, and the development of the comet assay was a result of this desire. I always felt that, once seen, the secret of aging and its prevention could be found.

2. Scientific foundation in India

As a child, I thought that I would find the secret to aging and make my parents immortal, but I had no knowledge about research and no intention to pursue it. In July 1967, when I entered King George's Medical College (KGMC) in Lucknow, India, it was with the goal of becoming a family doctor in a village like the one that I had just left or a small town clinic. But KGMC was a unique place. Set on the Gomti River, it is a famously beautiful campus in a city known for its culture and courtliness. At the time, it was the top medical college in India, and its alumni, called Georgians, were top physicians, surgeons and researchers. It was also very well funded. I was exposed to new fields, taught by experts, and I had the opportunity to be in a lab. I stayed there for nearly ten years as a student, then post-graduate and finally as faculty.

During my post-graduate studies in the Department of Anatomy, I had the privilege of establishing a laboratory where I could study chromosomes under the microscope. My childhood desire to find the secret of aging was within my reach! I used to soak red kidney beans in water for 2 to 3 h, then blend and centrifuge them. I would remove the top supernatant layer using an ordinary pipet and syringe. This solution was rich in phytohemagglutinin and was used to stimulate human lymphocytes to divide. After using colchicine to arrest the cell cycle at metaphase, I could see a cell frozen in the midst of division. Finally, I had a chance to look at chromosomes, 46 of them. I ended up writing my thesis on chromosomal aberrations observed after treatments with hormones and antibiotics. During my Master's program, my supervisor, Professor Avinash Chandra Das, Chair of the Department of Anatomy, found funding to create a cytogenetics

☆ This article is part of the Reflections in Mutation Research series. To suggest topics and authors for Reflections, readers should contact the series editor, G.R. Hoffmann (ghoffmann@holycross.edu).

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1383-5742/

laboratory and I was only too eager to set it up. This was the beginning of my journey into DNA damage and aging research.

Our conditions were not perfect: the room was a converted processing area for anatomy specimens and body parts. We were missing some key equipment but we found substitutes—I took a pressure cooker from our kitchen at home and this served as the autoclave for our glassware. Without having fully sterile conditions, I used to lose 90% of my cultures to contamination. I had a UV light and a glass chamber that I sterilized using the light. I had a water bath, light microscope and electric centrifuge but no incubator. Electricity outages were common, almost everyday occurrences, and they interrupted many experiments. Still, by aspirating rabbit bone marrow directly, using colchicine to arrest cell division in metaphase, and staining with Wright's or Giemsa stain, we were able to visualize chromosomes. I found effects of antibiotics (tetracycline, chloramphenicol) but not of hormones (testosterone, estrogen and progesterone) on rabbit chromosomes after 7 days of daily injections [4].

Eventually, I wanted to see DNA, not just chromosomes, but this goal exceeded the resources and knowledge at KGMC. In the fall of 1977, I visited the labs of Drs. Geeta Talukedar and Archana Sharma in Calcutta to learn autoradiography and unscheduled DNA synthesis (UDS). The incorporation of radioactive bases into damaged DNA during UDS allowed for the estimation of repair in DNA by visual grain counting. In 1978, I traveled to Bhabha Atomic Research Center in Bombay to learn mutagenesis in bacteria—the Ames test—with Drs. A.S. Aiyar and P.S. Chauhan. This allowed me to quantify the number of mutations induced by environmental chemicals. Still, even at Bhabha, they were not studying DNA damage directly. By the time I left Bombay, I had the notion that I would try to make an assay to directly measure a cell's DNA damage.

Wanting to work with DNA directly, I read any article that I could find on DNA damage, sister-chromatid exchange (SCE), alkaline elution and chromosomal aberrations. The medical library at KGMC had few scientific journals, so I used to read articles in the well-stocked archives of the National Botanic Garden and Central Drug Research Institute, both in Lucknow. On countless occasions, my wife would copy the articles by hand so that I could read and replicate experiments in the lab. After I had left Lucknow and arrived in America, I showed these handwritten copies of articles to their original authors. Ronald Hart was incredulous and amusedly took these papers around the labs at the National Center for Toxicological Research (NCTR). Painstakingly copied in blue ink were the articles of Drs. Nathan Shock, Ed Schneider, George Martin and Dr. Hart himself. I gained a lot of knowledge from this published work, and it inspired me toward new research directions and even life style changes. While I was still in India, Lester Packer's work on vitamin E's effect on WI-38 cells, making them immortal [5], inspired me to buy a bottle of vitamin E oil for daily ingestion.

3. Research training in the United States

Having taken advantage of all the resources available in India for studying DNA damage, I began to look for a post-doctoral fellowship. I wrote letters to every author outside of India whose work I had read and respected. Two positive responses came: one first from Dr. Hart and then one from Dr. Ed Schneider. I accepted Dr. Hart's offer as he was more of a basic researcher. The airplane ticket was equivalent to six months of my salary as a demonstrator in KGMC's anatomy department, where teaching medical students was my main job. I had to borrow money from my father and a fellow "Georgian," the co-author of my first publication, Dr. M.K. Tolani. I had never left India before, but a month after Dr. Hart's letter arrived, I traveled 12,500 miles—exactly half way around the earth.

I arrived at Ohio State University on the 10th of October, 1979, as a post-doctoral fellow. I had less than a hundred dollars in cash, a letter from Dr. Hart, and a suitcase filled with cashew nuts and raisins. As a vegetarian, I had no idea what I would find to eat in the United States. I was fortunate to have the best possible guide into American life; like a kindly grandmother, Mrs. Helen Dixon hosted many foreign postdocs in her large home near campus. She was my good friend and host for my entire time at OSU. My supervisor and the head of our lab, Dr. Hart was tall and vibrant with a booming laugh that conveyed positivity and progress. My main project at OSU was studying the effects of known carcinogens in rat tissue. The animals were sacrificed to estimate DNA damage in various organs. My approach was initially limited to mincing the organs with scalpels in a crisscrossing motion on frosted glass to get single-cell suspensions of the tissues that were then used for a variety of assessments. I spent many contented hours in the lab. I emerged to use OSU's playing fields and swimming pools, trying American-style football, diving or tennis. Many weekends were spent in the immigration offices of Cincinnati, where I struggled to obtain a temporary or permanent status that would allow me to stay in the country.

As I was finishing my postdoctoral fellowship at OSU, I was offered a position in Jefferson, Arkansas, in January of 1981. Dr. Hart had been appointed director of the NCTR, and he asked me to be part of his team. He had ambitious goals. Alongside Drs. Ming Chang and Angelo Turturro, I worked on assessing the effects of asbestos in vivo and in vitro [7]. I also developed a novel technique to infuse BrdU using an intraperitoneal catheter *in utero* in rats [8]. We then found stage-dependent effects of toxic agents on fetal development by studying SCEs in various tissues in embryos at various stages of development [9].

4. Formative ideas for the comet assay

When my appointment as a visiting scientist at NCTR ended, Dr. Steve D'Ambrosio offered me a position as Visiting Assistant Professor back at OSU in 1982. Returning to OSU resulted in my long-lasting research collaboration and friendship with Dr. Ralph Stephens. I learned more about staining DNA working with Dr. Stephens than I had ever known and that was a starting point for developing a new technique. We even published a methods paper showing differences in staining between live and dead cells [10]. By this time, I was familiar with several techniques for assessing DNA damage, including the alkaline sucrose gradient technique, which I had learned from Dr. Hart, and the UDS assay. As a postdoctoral fellow, I also became proficient in the nucleoid sedimentation technique, thanks to the guidance of Drs. Philip Lipetz and Ralph Stephens. In this technique, the nonionic detergent Triton X-100 was added to a high salt (2.5 M) solution for rapid lysis of cells.

Learning these techniques and knowing their drawbacks laid the foundation of ideas for a new technique. While I was still a postdoc, Dr. Douglas Brash, by chance, gave me a book chapter by Rydberg and Johanson [6]. Rydberg and Johanson's technique involved embedding lymphocytes in agarose gel, lysing cells with a solution of detergent (SDS) and EDTA on microscope slides, air drying cells in agarose, treating with an alkaline solution, and then immersing cells and gels in a neutralizing solution before staining with acridine orange. I studied the work overnight, and the next day Dr. Brash told me how to make agarose, mix it with the cells and solidify it on microscope slides. In this technique, the alkaline solution unwinds the DNA, which, after staining, appears as a halo in damaged cells. The intercalation of dye in double-stranded DNA is responsible for the green fluorescence, and the red fluorescence is due to the association of acridine orange along the single stranded DNA. Quantification of the ratio between green and red

was done with a special microscope that measured their intensity. The technique estimates DNA damage using a ratio of green to red fluorescence; it cannot quantify the number of DNA strand breaks, but it can be used as an index of DNA damage. However, the variability was so great that I could never properly visualize or assess induced DNA damage. I repeated the technique to the point of exhaustion, but the results seemed to be pH-dependent, concentration-dependent and time-dependent. I spent many hours in the zoology labs at OSU because Dr. Hart's lab, with its focus on alkaline sucrose gradient, had no fluorescence microscope. I liked the idea of embedding cells in agarose, but I still wanted a way to directly quantify DNA damage.

In May of 1982, I attended the First World Congress on Toxicology and Environmental Health in Washington, D.C. At the poster session of my work, I saw Dr. Raymond Tice. I was surprised to see his name-tag, and I asked him, "Are you the same Tice?" He smiled and said, "Yes, I'm the same Ray Tice." Incredulous that the man whose work I had read for so long would be visiting my poster, I asked again and got the same answer. Dr. Tice had been a Ph.D. student under Dr. Schneider, and we had common research interests. Thus began our collaboration. We exchanged phone calls and letters, and over the next ten years we would publish several papers [11–17], beginning with the 1988 paper that forms the basis of what is now known as the comet assay.

5. The path to the comet assay

In 1985, for several months after my appointment at OSU ended, I was jobless and I spent the time thinking of the ideal technique to assess DNA damage. I already knew I would embed cells in agarose as Rydberg and Johanson had done. At that time, I realized that I had three problems: isolation of living cells, embedding of cells, and lysis of cells. During this otherwise infertile, idle period, the idea came to me to electrophorese the cells in order to move the small, negatively-charged DNA pieces outside of the nucleus. Frustratingly, I had no lab or resources to test it. In a lucky stroke, Dr. Schneider called me from the University of Southern California (USC) in the fall of 1985 to tell me that he was going to the National Institutes of Health (NIH), and he asked me to join him there in the National Institute on Aging (NIA).

Dr. Schneider wanted someone in his lab to be trained in alkaline elution. He had found a perfect place and so for the last two months of the year, I went to Lausanne to learn alkaline elution in the laboratory of Dr. Peter Cerutti at the Swiss Institute for Experimental Cancer Research. Dr. Cerutti was a thorough teacher. At the end of my visit, he gave a dinner for me at his house. He had a spread of cheeses, breads and special foods. He offered me a spoonful of something very shiny, gray-white in color. He put it directly on my plate and I promptly ate it, inquiring only after it was in my mouth what it was. Caviar, he told me. I kept chewing and asked, "What is caviar?" Fish eggs, he replied. As a vegetarian, I was horrified and had to ask for the restroom! Dr. Cerutti was equally horrified. He thought he was offering me a real treat! What I learned in the lab, however, was an inspiration for me, and Dr. Cerutti would later make several visits to NIA to see our progress. I must have spoken of him often at home because my young daughter, when given a little yarn doll as a gift, promptly named it Peter Cerutti.

From Switzerland, I went back to NIA and published a paper on alkaline elution of sperm [12]. Still, I could see drawbacks to the alkaline-elution technique: it could have up to 30% variation in the same sample, even with the same cells under the same conditions. Although I was not satisfied with the technique, I did pick up the idea that sorting DNA according to molecular weight was viable and could be informative. Even while setting up Dr. Schneider's lab for alkaline elution in 1986, I remained eager to start working on the idea of alkaline microgel electrophoresis. I did many experiments applying current to cells in agarose, but I was not able to get rid of RNA or get the right resolution. Slowly, I was refining the method. I made microgels after isolating lymphocytes, lysing the cells in high salt with two detergents, and doing electrophoresis in highly alkaline solution. Lacking samples during these early days of development, I used my own blood, sometimes pricking my finger several times a day. I thought to precipitate the DNA after lysis and electrophoresis because localized DNA could be detected and measured more easily. I worked on precipitating DNA using ammonium acetate and ethanol combinations, spermine and ethanol combinations, and later, cetrimonium bromide (CTAB) to precipitate small amounts of DNA. I then washed the DNA in ethanol and dried the slides. In previous attempts, I had used a neutral solution with acridine orange. Now I tried an alkaline solution of ethidium bromide. It proved to be the most stable and sensitive.

I was gaining more knowledge about the structure of DNA under neutral and alkaline conditions, and I thought it would be more sensitive to use alkaline electrophoresis. As a bonus, RNA is degraded under alkaline conditions. The conditions also denatured DNA, revealing the breaks. I could easily see damage from X-rays, and for the first time I saw comet-like images with a streaming tail rather than a faint break here or there. I could not believe it! I was jubilant to see the tail, which I knew signified DNA (not RNA). I ran to tell everyone in the lab: Mike McCoy, Dr. Tice and Dr. Schneider. They had some concerns about whether the technique could be reproduced, and I started new experiments straightaway. I succeeded in showing a difference between controls and cells treated with 200 rads (2 Gy) of X-rays, but the goal remained to make the technique sensitive enough to detect damage caused by

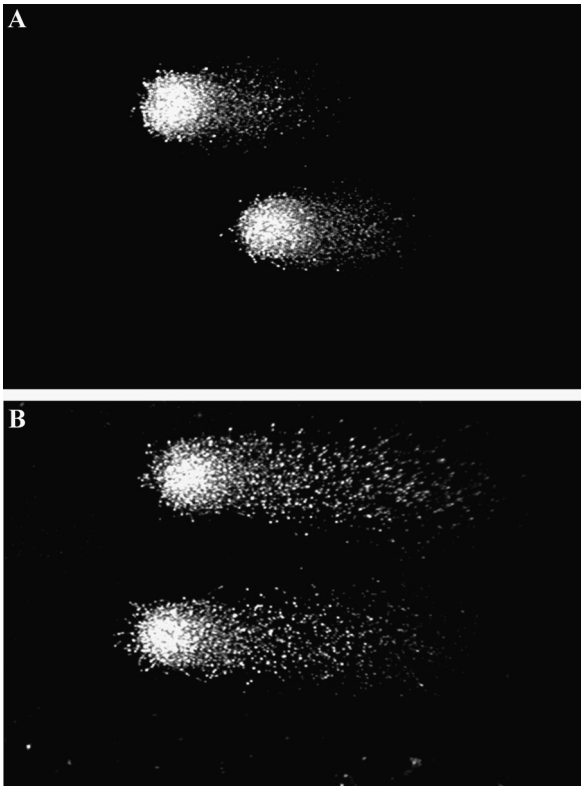

Fig 1. Comet assay. (A) shows two human leukocytes, representing an untreated control after single-cell gel electrophoresis. (B) shows two human leukocytes that had been irradiated with 100 rads (1 Gy) of X-rays in one minute. The comet-like tail consists of small fragments of DNA that arose by DNA strand breakage (dye: YOYO-1; magnification 400x).

25 rads (250 mGy) of X-rays. Taken from these early experiments, Fig. 1 shows control and irradiated human lymphocytes after microgel electrophoresis.

When I had completed a draft of my manuscript, Dr. Tice, who often came up from Integrated Laboratory Systems in Research Triangle Park, NC, to visit NIA, informed me that Ostling and Johanson had published similar work a few years earlier, in 1984. I went to the library soon after the meeting to read their paper. Ostling and Johanson [18] had added a novel step, electrophoresis, to the Rydberg and Johanson technique described earlier. Their new method, however, had two major disadvantages. First, due to the significant amount of RNA, estimation of the correct amount of DNA was not possible. When high quality agarose is properly made and layered with sufficient thickness on top of a layer of cells, the matrix retains DNA strands, RNA and small, broken fragments of DNA. I wanted to see DNA strands and broken pieces of DNA but not RNA. Second, sensitivity was limited by the conditions used for dissociation of the chromatin, which allowed DNA to maintain its tertiary and quaternary structures. Ostling and Johanson had used a neutral solution for cell lysis. DNA, with tertiary and quaternary structure intact, does not move in a predictable manner.

In the work that we were about to submit for publication, we had electrophoresed lysed cells under alkaline conditions to partially disrupt secondary structure and to remove the DNA's tertiary and quaternary structure. This allowed more predictable movement of DNA in the agarose. Alkaline conditions also degrade RNA and reveal more DNA lesions, including single-strand breaks, double-strand breaks, alkali-labile sites, etc., so they are more sensitive than neutral conditions that reveal only double-strand breaks. This is the basis of the comet assay's sensitivity. Ostling and Johanson were unable to detect less than 100 rads of damage, while we had detected significant changes at 25 rads. Finally, Ostling and Johanson had stained DNA with acridine orange and used a fluorescence ratio calculation at two points (nucleus and tail) as an index of DNA damage rather than migration distance. For these reasons, I knew that the technique that we were about to publish would be unique and sensitive. Some years later, after our publication of the 1988 paper, Dr. Karl-Johan Johanson came to my lab at the University of Washington with his colleague Dr. Britt-Marie Svedenstål to see the kind of research we were doing. He was a man of few words, but he was kind and tolerant and showed a true love of science.

6. Applications of the comet assay

Our 1988 paper on this technique [11] was, I felt, a big step in the right direction. My goal had always been to develop a technique to visualize aging but my larger aim was to elucidate the causes and mechanisms of aging. At this point, I integrated my original aim with the new technique. I thought that maybe the technique would be sensitive enough to see changes caused by aging. Using blood samples from NIA's Baltimore Longitudinal Study on Aging, we compared DNA damage levels in young and old individuals and found significant differences [13]. For the first time, I was able to observe changes in the DNA of a single cell due to aging. This had been the driving force behind my leaving my home institution in Lucknow, and I felt I had finally found my path.

I was thrilled by seeing the evidence of aging but the relationship was not as overwhelming as I had hoped, and I wanted to do a better study with more samples and different cell types. I thought of more experiments. It occurred to me that sperm should not be aging and that there should be zero damage. I looked at other cell types that, like sperm, had condensed chromatin and I found that chicken erythrocytes would offer similar condensation. So I drove from Baltimore to a farm in rural Maryland to get some fresh chicken blood. After finding extensive DNA breaks, we theorized that alkali-labile sites are a characteristic of condensed chromatin [14,15]. This was confirmed when we compared levels of DNA damage in mouse and human sperm [12].

Perhaps because I now had a newborn at home, one phenomenon particularly interested me: two adults, with relatively old cells, can produce a new baby with perfect, intact DNA. How does this happen? After seeing how many breaks were present in sperm cells, I speculated that the breaks could be repaired by meiotic proteins before fertilization in order to produce healthy new offspring. I became interested in recombinational repair and was particularly interested in the work of a Japanese scientist, Dr. Yasuo Hotta, who had isolated a recombinase protein. I wrote to Dr. Hotta to ask whether I could visit his lab to learn more about recombinases. He responded favorably and was kind enough to suggest a source of support. Through the generosity of the Japanese Society for the Promotion of Science, I was able to stay in Japan for two months. This was a wonderful experience both in the lab and outside of it. Dr. Hotta, his team and Dr. Takahiro Kunisada were ideal hosts, and I went away with friendships, a great deal of knowledge and some new ideas.

In 1989 I left NIH to be with my wife and young children in rural Washington State. At nearby Eastern Washington University, I continued to do DNA damage research [16,17], explored the relationship between DNA damage and disease, and observed DNA damage in an Alzheimer's model cell line. As an adjunct professor, I had a lab but no salary or budget for supplies or equipment. I wrote several unfunded grant proposals on aging, and after a year I was looking for a new position.

In 1991, with the help of Dr. Schneider, I moved to USC where I performed modifications of the technique (e.g., trypsinized and nontrypsinized cells) with various kinds of agarose (e.g., low melting point but high resolution). None of the adaptations provided enough sensitivity. My goal was to detect the minute changes of human life: exercise, X-rays, even deep inhalation. We made several technical modifications to further enhance sensitivity [19]. To free nuclear DNA of proteins, we introduced a proteinase-K step that could be applied after or during regular lysis. To apply a uniform electric field, which minimizes variation in DNA migration from cell to cell and slide to slide, we modified the electrophoretic unit and used a recirculating antioxidant-rich alkaline electrophoretic solution. I tried many different kinds of dyes that might make the technique more sensitive. I used to go around the nearby labs, looking to get a few drops of any unusual dye – anything I could get my hands on – "Are you using that? No? Can I borrow it?" Anything that I could not find, I ordered from the Sigma catalog. I tried 21 different dyes before settling on YOYO-1, an intense fluorescent dye that detects electrophoretically migrated DNA extremely well. These changes enabled us to detect significant DNA damage at doses as low as 5 rads (50 mGy) of gamma-rays [19].

I then wanted to see whether the assay could detect the effects of an extremely-low frequency (60-Hz) field. My family was now in Seattle, so I telephoned researchers and department heads at the University of Washington (UW) trying to find someone studying the effects of extremely low-frequency radiation. Dr. Arthur Guy, who was head of the Bioelectromagnetics Research Laboratory, referred me to Dr. Henry Lai. Dr. Lai told me that it was unlikely that a 60-Hz field could affect DNA because its energy level was so low, but he proposed that we look at radiofrequency radiation because its energy is higher. Enthusiastic about this possibility, I decided to leave USC and work with Dr. Lai without pay until we could secure funding. In 1994, we finished our first experiments. I prepared slides and flew with them back to USC to perform the analysis because we still did not have a fluorescence microscope with image analysis at UW. Using the comet assay, we were able to detect

increased DNA damage in brain cells of rats exposed to radiofrequency radiation at as low as 0.6 W/kg. The standard for cell phones in North America at that time was a maximum of 1.6 W/kg. The experiments with Dr. Lai on the effects of electromagnetic fields [21] and radiofrequency radiation [22] were the beginning of my longest scientific partnership, and Dr. Lai became both friend and mentor in my new environment at UW. On the basis of our studies on radiofrequency radiation, we obtained funding to do further studies and found that 60-Hz fields caused DNA damage [23–25] at a similar frequency to that used by cell phones. Unintentionally controversial, our findings were regarded as a challenge to the growing cell phone industry.

In 1995, we introduced the use of ethanol precipitation of migrated DNA in agarose to enhance the sensitivity of detection of DNA in microgels. This method also allowed slides to be preserved for future use. Our experimental design for these studies was simple. I taped microfuge tubes of lymphocytes to a wooden ruler at the 5, 10 and 20 cm marks. I placed the ruler next to a gamma ray source (technetium-99) and the data showed a clear dose response relationship [20]. Exposure at 4°C prevented DNA repair, resulting in unmitigated accumulation of DNA damage for the duration of the exposure. We were able to detect a significant increase in DNA single-strand breaks at a dose as low as 1 rad (10 mGy).

I also wanted to use the technique to study the effects of various common substances. Alcohol works as an antioxidant in leukocyte cultures and does not cause DNA damage; the story is very different in vivo where ethanol is metabolized into toxic acetaldehyde. In our work, we intubated rats with alcohol and dissected their brains to find the time kinetics of DNA damage. We found significant DNA damage from ethanol [26]. We also observed that the metabolite acetaldehyde is genotoxic [27] in human lymphocytes in vitro. I then thought that the same substances or experiences can be oxidant (damaging to DNA) or antioxidant depending on the existing defenses of an individual. I investigated the effects of antioxidants, such as vitamin C, on human lymphocytes, human diploid fibroblasts and MOLT-4 human leukemia cells and found a significant DNA damaging effect from moderate doses of sodium ascorbate [28].

At this point I felt the assay was sensitive enough to detect the minute changes that lead to aging and simple enough to be a regular part of my routine. In fact, I had incorporated the technique into my daily life. I would make small changes in lifestyle and test their effects; I did the comet assay on myself almost every day, after playing tennis, swimming, eating half a dozen carrots or trying a new vitamin regimen.

In 1995, a collaboration allowed us to see beyond the number of DNA breaks: Dr. A. T. Natarajan at Leiden University, an expert in chromosome hybridization, led a study combining the neutral comet assay with the FISH technique. This successful combination of techniques allowed us to see genes, centromeres and telomeres, and we were able to visualize the location of gene segments. For the first time I could see specific genes in the halo of the comet, where we identified the O^6-methylguanine-DNA methyltransferase gene [29].

After working for so many years with human chromosomes and DNA, in 1999 I directed my research toward bacteria. There were two reasons: (1) I wanted to know whether replicating *Escherichia coli*, having a theta (θ) shaped chromosome, would have one straight chromosome if broken. Only one double-strand break would be needed to do this and therefore, (2) I wanted to know the sensitivity of detecting only one double-strand break for testing antibiotics or chemicals. Neutral conditions were used to reveal only double-strand breaks, which are lethal in bacterial cells. The neutral comet assay revealed a simple and elegant demonstration of these breaks: an *E. coli* nucleoid with a single tail of DNA streaming behind it [30].

Our next iteration of the comet assay was only peripherally related to DNA damage: a sensitive method for visualization of apoptosis on a cell-by-cell basis. In the DNA diffusion assay [31], cells are lysed in alkaline detergent solution, embedded in agarose, and stained using my very favorite dye, YOYO. The technique also takes advantage of the numerous alkali-labile sites in DNA of damaged cells. Under alkaline conditions, these fragments of DNA diffuse outward from the nucleus and give apoptotic cells the appearance of a halo. Studying apoptosis, I realized, was crucial in studying how damaged cells are eliminated and thus, critical to studying healthy aging.

The versatility of a technique lies in its adaptability to a variety of tissues. Using the comet assay in collaboration with Dr. Norman Wolf of the Department of Pathology at UW, we were able to show increasing DNA damage with age and with light exposure in lens epithelial cells [32],which Dr. Wolf showed was related to cataract formation [33]. We also used an innovative method of dispersing a variety of tissues into single cell suspensions, including the kidney (one of the hardest tissues). Dispersion of tissues into single cells is required in many biological assays but the procedure often causes damage (e.g., the mincing method that I used as a post-doc!), and there was a need for a device to minimize DNA damage while still effectively dispersing tissue. I had earlier worked with a gentleman named Tim Hopkins, who designed a specialized and novel system, the Tissue Press [34]. A few years later he called me up with an unusual offer. He had a new device which was intended for use in immunizations and he wondered if this device, the Biojector, could be adapted for use with the comet assay. The CO_2 cartridge, which was the source of pressure in the syringe, rapidly dispersed any tissue into single cells through a narrow (<50 micron wide) hole with minimal procedural damage. Using this dispersion method, in 2001, we were able to show an increase in DNA damage with age in mouse kidney cells in collaboration with Dr. Wolf and Dr. George Martin. Dr. Martin was the first to correlate lifespan with cloning efficiency in the rat model [35] and one of the authors I looked up in the libraries in Lucknow, India. We were also able to quantify and calibrate this increase with such sensitivity that we could show the equivalent of 12 months of aging in terms of rads of X-rays and number of DNA double-strand breaks [36].

Yet, I still had not answered critical questions about the aging process. I had tried to assay DNA damage in human sperm since I had first developed the assay. No matter how much I tried, it did not move during electrophoresis. Even after 24 h and 400 rads or more of X-rays, I saw no DNA migration. Searching the literature, I read that sperm chromatin was highly condensed. The process of chromatin condensation requires crosslinks between DNA and proteins, such as protamines but also some histones. Using Proteinase-K in lysing solution to decondense chromatin finally allowed me to see an X-ray dose response in sperm exposed to radiation. In 1997, Dr. Stephens and I had introduced a neutral version of the assay to detect X-ray induced DNA damage in human lymphocytes [37]. In 1998, we used this neutral version of the assay to detect DNA double-strand breaks in sperm cells [38]. This neutral comet assay, using proteinase-K, sensitively detected DNA damage in sperm and I continue to use it in a variety of studies. For example, with Drs. Bhaskar Gollapudi and Sue Marty, we were able to show a relationship between p53 and levels of DNA damage in mouse sperm [39]. In collaboration with Dr. Charles Muller of the UW's Male Fertility Clinic, we showed a significant increase in DNA damage and a surprising decrease in apoptosis after the age of 35 [40]. This meant that men older than 35 had sperm with high levels of DNA damage that would not be eliminated by apoptosis and might go on to fertilize an ovum. This finding, labeled a "male biological clock," attracted high levels of scientific and media interest. For me, our work contradicted my earlier theory that gametes repair their DNA damage before

fertilization. Our findings led to new research directions that I would still like to pursue, specifically the fetal origins of adult disease.

Many researchers, including myself, had by this time shown relationships between mutagens and DNA strand breaks using the comet assay. However, my real work in environmental chemicals and DNA damage began with my collaborations with Dr. Russ Hauser at the Harvard School of Public Health who was principal investigator on a large study of phthalates (a class of chemicals found in a variety of household plastic products). Our ultimate goal was to study the effects of phthalates, PCBs and insecticides on sperm DNA. We found that urinary levels of these chemicals were associated with increased levels of sperm DNA damage [41]. Other studies with Drs. Hauser, Susan Duty and Zuying Chen investigated the comet assay in relation to fresh and flash-frozen semen samples [42], semen parameters [43] and insecticides [44]. A collaboration with Dr. John Wise [45] on environmental and occupational exposures to chemicals also contributed to toxicological applications of the technique. Several CDC and NIOSH studies have recently used the comet assay to study occupational exposures. In collaboration with Dr. Mark Toraason, we found increased DNA damage in the leukocytes of factory workers exposed to spray adhesive chemicals, such as bromopropane [46]. In collaboration with Dr. Mark Boeniger, we studied polycyclic aromatic hydrocarbons (e.g., benzopyrene; dimethylbenzanthracene) and DNA damage in auto repair workers. These studies prompted me to develop a protocol for the collection of samples in the field, their storage, and their shipment from the agency conducting the study (in our case, CDC and NIOSH) to a laboratory for freezing, thawing and assessment of DNA damage. This protocol was used by the CDC for a project headed by Dr. Mary Ann Butler to study workers exposed to Jet Fuel at US Air Force bases [47].

7. Refinement and new directions for the assay

Real refinement of the comet assay came through customization of the equipment. After experimenting with the electrophoretic units used in other techniques, in the mid-1990s I decided to make my own. In consultation with Ralph Stephens, I began to design a specialized unit. Early on, I would saw flat sheets of Lucite and glue them together in order to realize my designs but they had problems due to their inexpert construction. We found a skilled manufacturer and designer, Clive Ellard (Ellard Instrumentation). The new unit solved some of the recurrent problems in the technique and allowed greater sensitivity. I then started to modify slides, because frosted slides caused background with YOYO dye. We had used frosted slides for better attachment of agarose, but the uneven background from the frosting made it difficult to analyze the migrated DNA using an image analysis system. Two changes were made to address this problem: the use of a tray to simultaneously process eight slides and the use of newly designed slides with a clear window and frosted borders [30]. These changes enhanced the sensitivity of the technique to the point that we could visualize an individual DNA double-strand break in *E. coli* [30].

Finally, I have worked to attain ultimate sensitivity for assessing the extent of DNA damage. Considering the comet as only a head and tail may be simplistic. I had to consider the comet in three parts: head, body and tail. The body consists of relaxed loops of DNA, and the tail consists of broken pieces of DNA. Our latest refinement of the comet assay is designed to retain these broken pieces of DNA. The earliest comet assay studies used a single parameter: comet length. However, the most complete picture of DNA damage is offered by the inclusion of a variety of parameters. Dr. Peggy Olive developed the parameter "Tail Moment" to assess intensity of broken DNA fluorescence. We developed the parameter "Integrated Intensity" to account for the three-dimensional aspects of DNA migration. I have worked to incorporate such parameters in computerized image analysis programs. I once had to rely on my own macros and a camera hooked up to a fluorescent microscope and computer. Now a variety of advanced image analysis systems have been developed and a reliable, automated system for use in labs and clinics is on the horizon.

8. The comet assay comes of age

The comet assay has been modified, adapted and adopted for various purposes over the past 25 years. Even the name has changed through the years. Ostling and Johanson [18] called their technique "Microelectrophoresis." In our 1988 paper [11], we named the assay "the Microgel Electrophoresis technique." Soon after the publication of this paper, I was invited to North Carolina to help set up Ray Tice's lab at Integrated Laboratory Systems. Dr. Tice, his versatile and gentlemanly technician Paul Andrews, and I came up with a better name. We called the technique Single Cell Gel Electrophoresis or just Single Cell Gel (SCG). Shortly afterward, Dr. Peggy Olive and colleagues introduced the term "comet assay" [48], and that has rightly stuck for the last 25 years.

In this span, researchers have applied the comet assay to a variety of fields. Dr. Andrew Collins and colleagues introduced the assay's use in human biomonitoring, studying the possible amelioration of DNA damage by nutritional supplements [49] and repair enzymes such as endonuclease and formamidopyrimidine DNA glycosylase [50]. Dr. Awadhesh Jha and others have innovated ecotoxicological applications of the assay for use in wildlife and environmental monitoring [51]. My early collaborator, Ray Tice, has taken the lead, along with Drs. Diana Anderson, Emilio Rojas, Yu Sasaki and others, in validating the assay's use in genotoxicology [52]. There have been concerted and ongoing efforts to develop international standards for the assay, including those of the American, Japanese and European Centers for the Validation of Alternative Methods and principally of the European Comet Assay Validation Group. On the basis of work by these centers and the collaborative efforts of several international working groups on the comet assay, the Organisation for Economic Development and Co-operation (OECD) adopted test guidelines for the comet assay in 2014 (https://ntp.niehs.nih.gov/iccvam/suppdocs/feddocs/oecd/oecd-tg489-2014.pdf). The assay is now an accepted method for human biomonitoring according to FDA (http://www.fda.gov/downloads/Drugs/Guidances/ucm074931.pdf) and WHO guidelines. The comet assay has long been an online presence. An NIH list-serve group was established more than 20 years ago by Dr. T.S. Kumaravel (comet-assay@list.nih.gov), who brought knowledge of the assay to thousands of experienced and novice researchers. Dr. Alok Dhawan established an online repository of protocols, discussion and research related to the assay (http://www.cometassayindia.org/).

If the comet assay had a birthplace, it would be the labs of the National Institutes, the hub of basic science research in America. Yet, the reach of the technique has quickly expanded beyond these borders, and I have been able to observe its application in England, Hong Kong, Germany, India, Italy, and Korea. Seeing the technique used in many different kinds of labs was evidence to me of its simplicity and an indicator of its future.

From arachnids [53] to zebra mussels [54], the comet assay has been used in plants, animals and microorganisms of all types. It has been applied to every kind of research that I could have imagined and at least one that I would never have imagined – precisely estimating the time of death in homicides [55]. This post-mortem application never occurred to me! My original impetus for the development of the technique was the study of aging and the

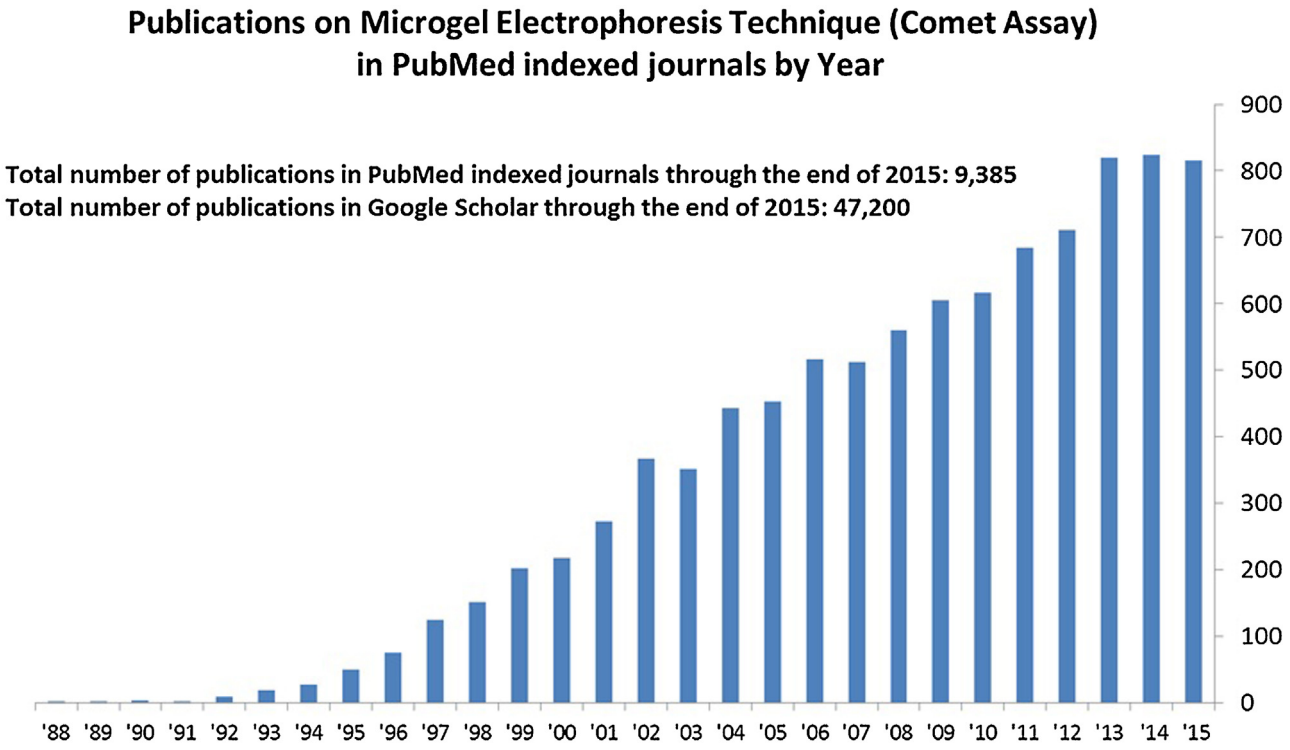

Fig. 2. Increasing numbers of publications using the microgel electrophoresis technique widely known as the comet assay. The numbers are publications in journals indexed by the National Library of Medicine's PubMed database since the description of the assay by Singh et al. in 1988 [11]. The search includes papers found using the search terms "comet assay," "microgel electrophoresis," or "single cell gel electrophoresis." Total numbers of publications are also shown for the exact search term "comet assay' in PubMed and Google Scholar.

extension of healthy human lifespan. I have worked mostly on studies in humans or animal models, but a variety of fascinating and significant research has been done in unusual organisms, wildlife and plants.

The past has been bright: the comet assay has detected DNA damage in a variety of organisms, tissues and cell types as a result of aging, disease and exposures. The recent emphasis on studying phenomena at the single-cell level will ensure its continuing relevance. As seen in Fig. 2, the number of publications using the technique has grown rapidly since 1988 and most rapidly in the last ten years. No other technique offers the same level of information in the same dramatic fashion: under the microscope we see those individual strands of DNA that form the basis of our existence, and we see their fragility as they break and trail out beyond their nucleus. It is a striking picture and one that is essential to understanding the health of our own species and a variety of others. As we develop ways to improve health and extend our lifespan, the future of the comet assay looks brighter still.

Conflict of interest

The author states that there are no conflicts of interest.

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