COMMENTS

Will an MRI Examination Damage Your Genes?

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Questions about possible genotoxic effects from magnetic resonance imaging (MRI) go back for many years, beginning with a (negative) study performed in 1980 by Wolff et al. (1). This issue has recently reemerged, with an inconsistent set of reports of DNA damage in human lymphocytes subsequent to cardiac MRI (Table 1). While evidence for DNA damage is reported in some published studies, in others it is not. According to the Organization for Economic Co-operation and Development (OECD), 107 MRI exams per 1,000 U.S. residents were performed in 2013 (2). Given the high number of MRI scans that are performed and the proposed use of high-field systems operating at field levels as high as 20 T (3), any evidence of possible genotoxic effects of MRI requires careful consideration. The technical issues involving these studies have been reviewed in detail elsewhere (4, 5). In this commentary, a broader range of issues are considered: 1. Is there a real effect (i.e., is MRI genotoxic)?; 2. What evidence exists that such effects might be caused by exposure to electromagnetic fields of some description?; and 3. The “so what” question: What is the potential significance to patient health of the effect, assuming it is real?

IS THERE A REAL EFFECT?

Table 1 shows a summary of recent (2007–) studies on genotoxic injury in humans exposed to MRI, or in human cells (mostly peripheral lymphocytes) exposed to MRI in vitro [see also Vijayalaxmi et al. (4) and Hill et al. (5)]. Studies were performed in vivo (using lymphocytes obtained from participants before and after imaging) or in vitro (exposing lymphocytes in MRI scanners). The majority of these studies used 1.5 T MRI systems (the most commonly used systems worldwide), and a few studies used 3 or 7 T systems. The studies varied in time of assay relative to exposure and assay type (most used the γ-H2AX assay). In addition, a few in vivo studies (not considered here) have been reported using rodents.

Overall, the studies listed in Table 1 are small, typically involving approximately 20 participants in total, or based on single blood draws from as few as one or two individuals. The reported effects have been modest in size [typically a doubling in double-strand breaks (DSBs)] and generally the increases have been only somewhat above normal biological variability, however, in some studies increases as high as 5 times above control values were reported.

The evidence is, at best, mixed. Some studies reporting increases in DNA damage [e.g., Fiechter et al. (6) and Lancellotti et al. (7)] are not supported by other, generally stronger studies [e.g., Reddig et al. (8), Fatahi et al. (9) and Brand et al. (10)]. The 2013 in vitro study by Szerencsi et al. (11) was specifically designed to validate the 2011 study by Lee et al. (12) and was unable to do so. The Brand et al. (10) in vivo study, in particular, was relatively large and well done; it was negative. However, the relevance of the negative studies to earlier positive studies has been challenged due to differences in design [e.g., in the time period the assays were performed relative to that of the MRI exposure (13)]. A considerable amount of additional work is required to resolve such disputes.

Apart from their small size, the studies frequently lacked obvious quality control measures, variously including blinding, sham exposures, concurrent controls and positive controls (see Table 1). One detailed critique of the work by Lancellotti et al. (7), published simultaneously with it, noted numerous technical weaknesses with the study (14). Perhaps the most serious limitation in Lancellotti et al. (7) was the lack of detailed follow-up of the participants subsequent to imaging, which prevented the investigators from addressing possible alternate causes of the increased number of damage foci observed in the later assays. The γ-H2AX assay detects damage foci associated with DNA repair, but is not a direct marker of DSBs (5); it can detect increases in damaged foci resulting from inflammation or infection (15) and even from aerobic physical exercise (16). The Lancellotti et al. (7) study was small and the statistical significance of the

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